Radiology Supplement

Contributed by

The American Osteopathic College of Radiology

These papers were presented at the
annual meeting in Los Angeles,
October 18-21, 1953

Volume 7
Number 1

August, 1954
Pages 1 to 18

RADIOLOGY SUPPLEMENT

The American Osteopathic College of Radiology

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Primary Pulmonary Neoplasms

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The term "primary pulmonary neoplasms" refers to tumors which originate in the bronchi or lung structure; these are of two general types, benign and malignant. Benign lung tumors, other than adenomas, hamartomas, and bronchogenic cysts, are very rare. They include fibromas, myomas, lipomas, chondromas, and neurogenic and mixed tumors.

BRONCHIAL ADENOMA

Of all lung tumors, adenomas are second in frequency only to bronchogenic carcinoma. They comprised 6 per cent of all lung neoplasms, according to the findings of Kramer and Som,[1] in a study of 355 bronchial tumors. Occurring in the early decades of life, they have a predilection for the left lung of the female.

They are probably derived from embryonic branchial rests or anlage in bronchial mucous glands or an admixture in ducts of the bronchial wall. They readily extend to peribronchial tissue.

Histologically, pulmonary adenomas are comprised of epithelial and mesodermal elements with a delayed growth index. Varying in size from a few millimeters to a dozen centimeters, they occur as dense, rounded, homogeneous masses, much of which is pulmonary and extrabronchial, or as pedunculated intraluminal growths. Their color varies from pink to cherry red. Encapsulation may exist.

The stroma of the tumor is highly vascular, loosely arranged, often suggesting endothelioma or angioma. However, with all the admixture of tissue, the delayed developmental course and therapeutic results lead to the belief that pulmonary adenomas are essentially benign, although the potentialities of malignancy cannot be ignored.

Symptomatology.—Cough, often dry, and chest discomfort, less severe than in cancer, are prodromal signs. The prominent symptom is recurring hemoptysis which may be exaggerated during menstruation. Early, the symptoms are often mistaken for hemorrhagic bronchiectasis. Later, dyspneic attacks due to bronchial stenosis may be ushered in. Progressively, with infection and pulmonary suppuration, cough becomes excessive, with profuse expectoration, which is often putrid in odor, fever, and increasing toxemia. Chronic bronchiectasis with empyema often intervening occurs late in the course.

Diagnosis.—Diagnostic signs seen in radiographs often result in confusing bronchial adenoma with bronchial carcinoma. The high vascularity and freedom from fixation or induration of the tumor is corroborative to the bronchoscopist. Ulceration is exceptional. Biopsy studies confirm the diagnosis.

Treatment.—Endoscopic treatment of the neoplasm if sufficiently early may result in its complete removal. However, this is exceptional. Destruction by diathermy is seldom feasible, owing to delayed discovery. In the event extension into adjacent peribronchial tissues has occurred, lobectomy or pulmonectomy is indicated. In view of the fact bronchogenic adenoma usually occurs during the early decades of life, surgical risk is reduced perceptibly. Leddy[2] states that this tumor is completely radioresistant.

PULMONARY CYSTS

Cysts of the lung vary greatly in size, arrangement, and composition. Except for hydatids they are very rare. Their content may be air, fluid, or solid matter. Air-filled cysts are the most frequent. Solid cysts, if congenital, are usually teratomas; more often they are dermoid or of parasitic origin (echinococcic).

Congenital cysts occasionally coexist with polycystic kidneys or cardiac malformations. They are usually multiple. If single, the dimensions are considerable.

The solid cysts or tumor masses may degenerate into cavity formation. Dermoid or intrapulmonary cysts are infrequently lined with gastric mucosa. A hemorrhagic cyst may result from contusion or laceration of pulmonary tissue.

Solid cysts are ordinarily recognized by roentgen studies, both direct and bronchographic. They appear as round densities with sharp outlines located peripherally.

HAMARTOMAS

Hamartomas are not true new growths but are tumorlike structures. They are comprised of an abnormal mixture of normal tissues. They may appear in any organ. In the lung they tend to contain an undue degree of fatty or cartilaginous tissue, hence are termed lipomas or chondromas.

Pulmonary hamartomas vary sharply in shape and size from the invisible to a tumor the size of a fetal head. The average is 1 cm. in diameter. The mass is often lobulated with well-defined margins and on the planigram has the appearance of a blackberry. No evidence of compression of pulmonary tissue is seen.

Cartilaginous and calcific plaques in the masses often are important diagnostic signs. A small lesion may simulate a Ghon tubercle or a metastatic nodule. Although hamartomas are classed as benign, the possibility of malignant change warrants their extirpation.

BRONCHOGENIC CARCINOMA

Introduction.—

The lung is one of the most common sites of primary malignant neoplasm, being exceeded only by the gastric cavity. Pulmonary cancer constitutes approximately 5 to 10 per cent of all malignant neoplasms.[2] It kills 15,000 Americans annually.

At Montefiore Hospital,[3] of 333 autopsies performed in instances of lung cancer, 260 or 78 per cent were in men, and 73 or 22 per cent were in women. Shanks and Kerley[4] and Bishop and Lindskog[5] give the ratio as 5 to 1.

In a series of 100 cases of primary pulmonary neoplasms reported in New Haven Hospital,[5] the average age of the patient was 55 years. However, young people are not immune. The incidence is about the same among white people and Negroes.[3,6] Moersch and Tinney[7] reported that cancer in the right lung is twice as frequent as in the left lung and that the lower right bronchus is oftenest included.

Authorities fail to agree as to causes of the increase in incidence of bronchial carcinoma; it may be due to an aging population and improved diagnostic and laboratory methods. Less confusion now exists in

differentiating these neoplasms from pulmonary tuberculosis, unresolved pneumonias, chronic lung abscess, and bronchiectasis. Mass chest surveys account for many of the current discoveries of lung tumors.

Etiology.—

The etiology of bronchial carcinoma is obscure. Only persons exposed to radioactive substances seem predestined to this involvement. Moersch and Leddy[8] in 1940 reviewed 250 cases at the Mayo Clinic and found no tangible etiologic factors. Simons[9] believes irritation is the real causative factor.

Alvarez[10] has given an opinion which is somewhat at variance with the above. He has stated, "I have read perhaps six statistical studies made here in America and abroad and I think it most probable that cigarette tar is the cause of the cancer."

Regato,[11] queried by me as to whether in his opinion smoking produced bronchogenic carcinoma, answered, "Almost 100 per cent; the few exceptions proving the rule; that is, inhalation smoking, such as cigarettes. Pipe and cigar smoking are much less responsible."

Origin.—

All primary lung cancers arise from cells in the basal layer of bronchial epithelium. The degree of differentiation determines the ultimate type.

In 75 per cent of the instances, bronchial cancer arises in a major bronchus,[3,6] usually near a bifurcation, resulting in the so-called central type of tumor. Early it may produce but a slight elevation of the mucosa and become irregular and fungated later. With the increase in size of the neoplasm the lumen is correspondingly reduced, causing obstructive emphysema of the pulmonary segment involved, with resultant atelectasis.

Upon penetration of the bronchial wall the tumor extends into the collapsed area and, distally, by way of the lymphatics, into various other parts of the lung. Coalescing, the neoplasm forms nodules and larger tumor masses, gray-white in color.

Early in the progress of the malignancy the hilar lymph glands are infiltrated and enlarged. By further spread through the lymphatics the tumor may reach the pleural surface, occasionally resulting in a hemorrhagic effusion.

Carcinoma of the main bronchus may result in shrinkage of the affected lung, giving the appearance of chronic pneumonitis with bronchiectasis.[3] In approximately one third of the cases, Fishberg and Rubin[12] found abscess formation, possibly the result of bronchial obstruction, or as is more probable, the tumor outgrew its blood supply, causing necrosis of its own tissue. This process occurs only in primary lung cancer.

Primary neoplasms originating in a small bronchus comprise the peripheral or nodular type. They include about 25 per cent of the cases.[3] These tumors are usually small, dense, and well-circumscribed and are often mistaken for metastatic lesions. At times the mass may involve an entire lobe; as such it is easily confused with pneumonia at the stage of consolidation. Later, the hilar lymph nodes are involved. On x-ray films, the tumor is seen separated from the hilus by a clear zone, a valuable landmark in the surgical evaluation.

Peripheral tumors are difficult to recognize, but the fact that they are highly malignant renders diagnosis especially important.

Symptoms.—

It is agreed that the initial stage of primary lung cancer is not discernible. The latent course may extend from months to as much as 2 to 3 years.

No classic or unerring symptoms prevail. Loss of weight and "indigestion" are often precursors to the diagnosis of neoplasm. The triad of cough, pain in the chest, and bloody sputum are perhaps the first to direct attention to chest disease. "Chronic bronchitis" is the term often applied to undiagnosed or unrecognized lung cancer. It can be easily confused with chronic tuberculosis in the aged.

The roentgenologic picture changes almost daily. Not infrequently the disease is first seen as an acute upper respiratory infection which may pass clinically as mild pneumonia (10 per cent).[13] This may result from a stenosed bronchus, producing first emphysema and then true acute pulmonary congestion. Often the initial symptoms are precipitate hemoptysis, occurring sooner or later in 40 to 50 per cent, or pleurisy, either dry or with blood-stained effusion. If bronchiectasis occurs the sputum is foul. A brassy voice suggests involvement of the recurrent laryngeal nerve.

Pain is variable, being severe in 50 to 60 per cent of cases.[13] It suggests invasion to bone or soft tissue, such as in Pancoast's syndrome. Loss of weight and weakness almost invariably occur as late symptoms. Wheeze or dyspnea, usually unilateral, will vary with the location of the tumor mass.

Tachycardia may occur as the result of involvement of sympathetic nerves. Cyanosis may occur suddenly or gradually, depending upon the degree of obstruction of the superior vena cava.

Diagnosis.—

It is generally conceded that no roentgenologic sign is definitely pathognomic of cancer of the lung. Early x-ray signs include unilateral infiltration of the hilus, a fan-shaped or segment-shaped zone of atelectasis, paralysis of the diaphragm, and a shift of the mediastinal structures toward the lesioned side.

Positive diagnosis is largely dependent upon microscopic examination of biopsy tissue or fluids.

With the bronchoscope, in 60 to 70 per cent of cases specimens can be obtained for biopsy study.[13] However, out of 147 cases described by Ochsner, DeBakey, and Dixon,[14] twenty-two peripheral lesions were not revealed by this method.

In the peripheral lobar type distal to the approach of the bronchoscope, bronchography is revealing. Appearance of malignant cells in the sputum, bronchial secretions, or pleural effusion is confirmatory. Planigraphy is of value.

In the event of failure of these methods, improved surgical technic, anesthesia, and antibiotics make diagnostic exploration of the chest justifiable.

Angiocardiography and pulmonary angiography have proved unique but valuable diagnostic procedures. Evaluating the relation of the tumor to the vascular system aids the surgeon in planning and conducting the operation.

Aspiration biopsy is condemned by some because of implantation of tumor cells in traversed tissue; Ackerman and Regato[6] state that they have never encountered any complications or seen tumor implantation in the tract of the needle.

Pathology.—

Histologically, almost all bronchial carcinomas readily fall into three classes;[3,5] (1) epidermoid

(squamous or basal cell), 30 per cent, (2) adenocarcinoma, 25 per cent, and (3) anaplastic or mixed, 40 per cent.

Fine differentiation of these tumors is probably impossible. Rather, they are often characterized by a mixture of various types of cells, the predominant ones determining the nomenclature applied. However, loosely stated, the squamous-cell or anaplastic neoplasms are located proximally in the lung while the small, peripheral tumors are usually adenocarcinoma.

Metastasis.—

Primary cancer of the bronchus tends to metastasize early and widely. Metastatic spread is facilitated by direct peribronchial invasion of a pulmonary vessel. Eighty to 95 per cent of the cases are complicated by lymphatic extension or true metastasis to other organs.[5] This percentage is greater than that found in carcinoma of any other viscera.

The site of distribution is practically unlimited, ranging in incidence from 72 per cent in the regional lymph nodes to 12 per cent in the heart and pericardium. This phenomenon is probably due to the continuous movements of the chest, including breathing and coughing, which disseminate tumor cells by way of the many large bronchial and vascular channels.

Treatment.—

Treatment depends upon the degree of involvement, that is, whether the tumor is operable or inoperable.

If malignant cells occur in the pleural fluid, or there is evident involvement of the phrenic nerve or brachial plexus, or metastatic deposits are found in distant areas, or the patient is far advanced in age, the case is considered inoperable. In a series of 412 cases in Charity Hospital in New Orleans,[14] 29 per cent were considered inoperable, while 71 per cent were considered operable. All untreated operable cases speedily became inoperable by extension. When a case is operable, pneumonectomy is the surgical choice. Of the cases submitted for surgery, favorable results are rare but encouraging; in the series of 412 cases the 5-year cure rate was 5 per cent. The present total number is still extremely small compared with the number of patients involved. With continued advances in chest surgery the outlook is rapidly improving.

Although bronchial carcinoma is generally considered radioresistant, radiation therapy has often a definite place in its treatment. All inoperable cases should be routinely treated by irradiation. To give symptomatic relief from pain in the chest, reduce cough, and diminish abnormal secretions it is doubtless of service. Atelectasis may be temporarily relieved by reducing the endobronchial tumor, and the patient's comfort is greatly although temporarily enhanced. If the tumor is highly malignant and metastasizes early, it is usually more radiosensitive.

The highly undifferentiated cells, including those of the so-called oat-cell tumors, are at first very radiosensitive but are unsuccessfully treated because of their rapid spread.

Generally speaking, patients should be sympathetically selected for radiation therapy. Undue enthusiasm should be carefully avoided to obviate a clinical end that may be worse than the original lesion.

Pancoast's Tumor.—

This tumor, which is also known as the syndrome of the superior pulmonary sulcus, was originally considered an extrapulmonary tumor of branchial cleft origin.[3] Now it is generally agreed by pathologists to be bronchogenic carcinoma with a predilection to peripheral extension and invasion.

Located high in the apex, it produces by pressure on the cervical sympathetics, soft tissues, and blood vessels severe pain at the base of the neck, upper chest, and areas supplied by the brachial plexus. Frequently a Horner's syndrome on the affected side results. Paralysis of the recurrent laryngeal nerve may result in hoarseness. Cough is usually absent.

Recognition by radiography is often possible. In the extreme lung apex and paravertebral region a dense sharply defined homogeneous mass is noted which frequently displaces the trachea and erodes proximal bone structures. Infiltrating the soft tissues the tumor superficially produces rounded nodules of varying sizes in the region involved.

Histologically, Pancoast's tumor may be squamous celled or anaplastic. Its progress is rapid and normally unrelenting. It is seldom amenable to successful removal or cure. Palliation in varying degrees may be accomplished by neurosurgery or high voltage x-rays. Its resistance to radiation varies somewhat with its cell structure, but it is generally considered quite radioresistant.

Primary Sarcoma.—

Some authorities state that the term "sarcoma" as applied to neoplasms of the lung is probably erroneous.[4] In a series of 5,600 autopsies in which 57 primary lung carcinomas were revealed, only a single sarcoma was demonstrated.[4] Lenk[15] discovered only one sarcoma in a series of 100 verified carcinomas.

This neoplasm may be round or spindle celled or a lymphosarcoma arising in the lymph glands or in the pulmonary or peribronchial connective tissue. The tumor is well defined, rounded, and nodular. It may be encapsulated. It varies greatly in size, occasionally involving an entire lobe. Cavitation rarely occurs. Bronchial obstruction is unusual. The pleura may be involved, occasionally resulting in hemorrhagic effusion into the pleural space. Metastases may develop, particularly to the liver. Treatment is primarily surgical.

Lymphangitic Carcinoma.—

This condition may originate in a small bronchogenic carcinoma; more frequently it is secondary to a cancer of other tissues.[3] It is generally a form of diffusely infiltrative carcinoma of the lung parenchyma and pleura by extension from a hilar lymph node.

The primary growth is usually insignificant in size, often microscopic. It diffuses lymph tissue in a weblike manner, the lymphatics appearing as yellowish-white streaks, variable in size, the result of plugging by tumor cells.

Physical signs and symptoms are not striking. Reduction in aeration space often produces dyspnea with associated anoxemia. Cyanosis may occur. Loss of weight and vitality suggests a generalized carcinomatosis with metastasis.

The roentgen findings reveal a reticular webbing representing an overlapping of pleural and pulmonary lymphatics. Fine striations intermixed with denser structures suggest sarcoidosis or hematogenous tuberculosis.

SUMMARY

1. The most common primary lung neoplasms, both benign and malignant, have been discussed.

2. The importance of radiography in identification has been pointed out. However, similarity of film

shadows of the various lung tumors renders differential diagnosis by roentgenography alone impossible. Acceptable diagnosis is by biopsy study of tissues and fluid examination.

3. Treatment is preferably surgical. In nonsurgical cases, irradiation therapy has a distinct place, largely as a palliative measure.

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Spinal Cord Tumors*

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The term "spinal cord tumor" is used to refer to any neoplasm within the vertebral canal. Less than one tenth of these (7 per cent)[1] are intramedullary, actually developing within the cord itself or occasionally invading it from without. Most spinal cord tumors are extramedullary and affect the cord or nerve roots by compressing them from without. These extramedullary tumors are designated according to their position as either subdural or extradural. About six in ten (64 per cent) spinal cord tumors are subdural; about three in ten (29 per cent) are extradural.[1]

Roughly 75 per cent of spinal cord tumors are primary in the vertebral canal, arising from the connective tissues covering the spinal nerve roots and cord or from the glial elements of the latter. The remaining 25 per cent of spinal cord tumors are secondary invaders of the vertebral canal. Of these, about one fourth arise as primary growths in the spine or paraspinal tissues; the other three fourths develop as spinal metastases from primary carcinomas in the breast, thyroid, prostate, lung, kidney, or gastrointestinal tract.[2]

It follows that characteristic radiographic evidence of vertebral involvement is to be expected with metastatic and primary spinal (vertebral) tumors which are producing neurologic symptoms by secondary invasion of the spinal canal. These changes sometimes permit radiographic identification of the nature of the tumor. By contrast, the primary spinal cord tumors, which originate outside the vertebrae, produce vertebral changes, if any, by secondary nonspecific pressure erosions of vertebral laminae, pedicles, or posterior body surfaces. These radiographic signs are indicative of the presence of tumor, but seldom diagnostic of its pathologic identity.

FREQUENCY AND DISTRIBUTION

Spinal cord tumors are about one-tenth as common as intracranial tumors and comprise approximately 0.5 to 1 per cent of all tumors. They occur in patients of all ages, about equally in both sexes, and in all regions of the spine. Their over-all frequency of distribution corresponds roughly to the relative lengths of different parts of the cord and to the relative number of cervical, thoracic, and lumbar vertebrae. Thus more tumors are found in the thoracic region than in the cervical, and still fewer in the lumbar and sacral regions. While this is true of spinal cord tumors as a group, certain pathologic types do show sites of predilection. The meningiomas and angiomas are found most frequently in the thoracic region, intervertebral disk herniations in the lower lumbar region, lipomas in the cervical and lumbar regions, and extradural cysts between the sixth and tenth thoracic vertebrae.[1]

SECONDARY SPINAL CORD TUMORS

Of the metastatic tumors secondarily invading the spinal canal I have already enumerated those primary carcinomas of various organs (breast, thyroid, prostate, lung, kidney, and gastrointestinal tract) which commonly metastasize to the vertebrae.

The more common primary tumors of the vertebrae which secondarily invade the spinal canal include chondromas, or intervertebral disk herniations, multiple myelomas, osteomas, vertebral angiomas (hemangiomas), and primary giant cell tumors.[2]

Chondroma (Intervertebral Disk Herniation).— Herniated disks, although not neoplasms as originally thought, should be considered in this connection because of their great frequency and because their symptoms are the same as those of tumor. Seven massive lumbar intervertebral disk herniations in my series have produced signs of cauda compression with complete or nearly complete oil block. At least one central type cervical disk herniation produced a syndrome of spinal cord compression previously mistaken for multiple sclerosis. It should be emphasized that the Queckenstedt test and myelography are indicated in suspected cases of multiple sclerosis where cerebral signs are lacking. Cervical cord compression may also simulate anterior poliomyelitis.[3] Herniated intervertebral disks will not be considered in this paper.

Multiple Myeloma.—

Myelomas occasionally arise primarily in a vertebral body, usually in the upper thoracic region. They

*Presented at the combined session of the American Osteopathic College of Radiology and the American Osteopathic Academy of Orthopedics, Los Angeles, October 21, 1953.

may remain solitary, or more characteristically metastasize as multiple lesions. Advanced destruction of vertebral bodies leads to their collapse with signs of root and cord impingement.[4] Presence of similar lesions in the flat and short bones and a clinical picture of anemia and cachexia are characteristic. Laminectomy may provide relief, but life expectancy is usually limited to about 2 years.

Case Report: A 44-year-old male, bedridden for 3 weeks with lower thoracic and upper lumbar pain and tenderness, complained also of nausea, vomiting, and loss of 30 pounds in weight. Five months before consultation an acute back strain occurred while lifting which did not respond to manipulation and physiotherapy. X-rays taken 2 months later showed angulation of the ninth and tenth thoracic and first lumbar vertebral bodies. Repeat roentgenograms showed multiple collapsed vertebral bodies and numerous scattered areas of circumscribed rarefactions in the skull and flat bones. Moderate anemia, increased sedimentation rate, albuminuria, and Bence-Jones protein were also demonstrated.

Diagnosis: Multiple myeloma.

Osteoma.—

Osteomas tend to arise from the vertebral processes rather than the bodies and may reach considerable size without causing root or cord compression, depending on their location. Roentgenographically the tumor is circumscribed with a dense cortex at its irregular periphery. Fairly normal trabeculae may be visible within the bony mass.[4] Relief of neurologic symptoms is often possible by laminectomy even with incomplete removal of the growth.[1] Vertebral osteomas causing neurologic symptoms are rather rare.

Case Report: A 57-year-old woman complained of recurrent low-back and left sciatic pain of 2 years' duration. Pain was exaggerated by coughing. Both Achilles tendon reflexes were absent and Lasegue's test was only slightly positive. The lateral aspect of the left foot was numb. X-rays showed a walnut-sized mass involving the left articular process at the level of the fourth disk. At operation excessive bone encroachment on the vertebral canal and intervertebral foramen was removed with relief of symptoms.

Diagnosis: Vertebral osteoma with caudal compression.

Primary Giant Cell Tumor—Primary giant cell tumors are genuinely malignant. They tend to arise in the vertebral arches, where they remain as solitary destructive lesions, usually confined to a single vertebra.[1] Radiographically these tumors are usually cystic, causing expansion of the affected part, often with coarse irregular trabeculae. Occasionally they are almost entirely osteolytic. Cord or root impingement may occur although vertebral collapse is rare.[4] Except when these lesions involve the vertebral body, surgical removal is often feasible. There is no tendency to recurrence. If involvement of the body precludes removal, roentgenotherapy may be beneficial.[1]

Hemangioma. — Vertebral hemangiomas will be described later as part of a special discussion of spinal angiomas.

Sarcoma—Primary sarcomas in vertebrae appear to be uncommon. Our experience includes only one such lesion which apparently arose from a vertebral lamina to invade the canal and extend extradurally from the third to the ninth thoracic vertebral levels. This tumor caused signs of cord compression and death at the age of 4 months in a female infant. Anaplasia prevented satisfactory subclassification of this primary mesenchymal tumor.

PRIMARY SPINAL CORD TUMORS

The most frequently encountered types of primary spinal cord tumors are nerve sheath tumors (26 to 29 per cent), meningiomas (23 to 25 per cent), gliomas, including ependymomas (10 to 13 per cent), and angiomas (3 per cent). Dermoids and epidermoids (less than 2 per cent), lipomas (less than 1.5 per cent), hemangioblastomas and chordomas are less common.

The primary spinal cord tumors are usually single, although exceptions may occur, especially in connection with nerve sheath tumors, as in Recklinghausen's disease, or neurofibromatosis, and with dissemination of some varieties of spinal cord glioma. Multiple intrathecal spinal implantations may also occur with intracranial gliomas, particularly with cerebellar medulloblastomas.[5]

Nerve Sheath Tumors.—The nerve sheath tumors (perineural fibroblastoma, neurofibroma, neurinoma, and the like) are soft irregular growths arising from spinal nerve roots. No special predilections for age, sex, or spinal regions are seen. Unlike the firmer meningiomas, they are rarely adherent to the dura, but in rare instances they may extend with the root through the dura to produce a dumbbell type of extradural and intradural tumor.[1] Diagnosis may often be established by roentgenographic visualization of the resulting enlargement of the intervertebral foramen. Oblique projections should be routinely made, in addition to the anteroposterior and lateral views, for study of the vertebral bodies, laminae, and processes.[6] The extraspinal portion of the tumor may be palpable in the neck or evident roentgenographically in the thorax or abdomen. Laminectomy and excision of the intradural portion should precede any attempt at removal of the extraspinal portion.

Meningiomas.—

Meningiomas are firm, rounded, fleshy growths, which are well encapsulated and seldom show any tendency to invade nervous tissue. They tend to produce a discrete depression in the cord which becomes angulated opposite the growth. Meningiomas are usually adherent to the dura and subdura, whether inside or outside the arachnoid, but are occasionally extradural.[1] Vascularity of the surrounding bone and soft tissues is generally increased. Predilection for the female sex and the thoracic region have been mentioned.

Meningiomas, as well as several less common varieties of tumor (ependymomas, epidermoids, and tumors of the filum terminale), occurring in the lumbar sac may reach relatively giant size, enlarging the vertebral canal and crowding the caudal roots. Surprisingly, symptoms with these caudal roots may be scant for a long period, but eventually low-back pain is followed by bilateral sciatica, paraplegia, sphincter disturbance, and sensory deficits in the saddle area.[4]

Case Report: A 60-year-old female complained of 2 years' gradually progressive difficulty in the use of the lower extremities. Weakness appeared first on the left followed by paresthesia; then paresthesia and weakness of the right lower extremity occurred. Pain was accentuated by straining. Sphincter control was retained. The colorless cerebrospinal fluid contained 120 mg. per cent of total protein. Myelography demonstrated oil block beginning at the seventh thoracic vertebra. At operation an olive-sized subdural meningioma was removed. Gradual recovery followed.

Gliomas.—

The preceding single, benign, encapsulated, and usually accessible primary spinal cord lesions (nerve sheath tumors and meningiomas) are, in a manner of speaking, a joy to the surgeon, for the results with surgical removal are usually gratifying. Not so, however, with the gliomas of the cord. These are sometimes multiple, usually intramedullary, and tend to be nonencapsulated and invasive. Many are absolutely inoperable. Elsberg[1] advocated dorsal incision and second-stage delivery after spontaneous extrusion for some types of well demarcated intramedullary lesions. Attempts at immediate removal are apt to increase the severity of symptoms by causing added damage to the cord.[6]

The more common varieties of spinal glioma include ependymoma, astrocytoma, glioblastoma multiform, oligodendroglioma, and medulloblastoma. Their histologic characteristics are identical with those of their intracranial counterparts which are more frequently seen.

Another lesion, although rare, which probably should be classed with gliomas of the cord is syringomyelia. Netter[7] illustrates the gross appearance of the cyst in syringomyelia which many authorities consider a result of necrosis of an intramedullary glioma. The resultant swelling of the cord is often demonstrated myelographically. Drainage by midline incision may lead to clinical improvement.

CORD TUMOR SYMPTOMS

Spinal cord tumors produce symptoms by mechanical irritation and compression of spinal nerve roots, compression and displacement of the spinal cord, and by obstruction of the vascular supply of these structures. As compression usually occurs gradually, the sequence in which symptoms appear varies with the position of the tumor relative to the dura and spinal roots and also with the spinal segment involved. Special monographs, such as Elsberg's,[1] consider these details of neurologic diagnosis which are beyond the scope of this paper.

Pain due to sensory root irritation is frequently an early symptom of cord tumor. Cervical cord tumors may cause pain in the neck, back of the head, or in an upper extremity. Tumors of the conus medullaris, cauda equina, and filum terminale produce pain in the low back radiating into the lower extremities and saddle area. Pain due to thoracic sensory root irritation is felt in an upper extremity or in the trunk where it may be mistaken for angina pectoris or the pain of acute visceral disease. The surgeon can avoid this error and unnecessary abdominal operations by using the jugular compression test. This is carried out by maintaining bilateral jugular compression until congestion of the face appears. Pain resulting from spinal nerve root impingement is intensified by the resulting shift of the fluid-distended arachnoidal sac covering the cord and spinal nerve roots. Pain arising from visceral disease is unaltered.[6]

With continued root compression signs of peripheral type motor involvement appear characterized by flaccid weakness and, eventually, atrophy. With progressive destruction of the nerve roots at the level of the tumor, a segmental sensory impairment develops and the initial symptom of pain may disappear. If pain is absent, its previous presence should be inquired into, as its location may be a valuable diagnostic sign.

Compression of the cord and interference with its circulation in the region of distortion impair transmission of sensory impulses from below upward past the level of the lesion. Cord compression also impairs conduction of controlling motor impulses downward past the lesion, thereby disturbing voluntary control of movement and sphincter action.

The degree of sensory loss will vary with the severity of cord compression. With lesser degrees of compression some sensory tracts can be more adversely affected than others, leading to dissociated sensory impairment (that is, greater loss of some sensory functions than of others), because tracts serving different functions are separated within the cord. But dissociated sensory disturbances are ultimately replaced by anesthesia, or loss of all forms of sensation, below the lesion as compression progresses. The upper level of sensory change, because of peculiarities of arrangement of sensory pathways within the cord and the vertical relationship between cord segments and vertebral levels, is usually several segments below the tumor. A zone of hyperesthesia can sometimes by delineated immediately above the upper level of anesthesia. When sensory losses first appear the upper level of change may be indefinite or variable and is likely to suggest a localization much lower than the actual site of the tumor.

Motor signs similarly vary with the site of the tumor. A tumor involving the cauda equina (made up of nerve roots below the lower end of the cord) will produce a flaccid paraplegia or lower motor neuron peripheral type flaccid paralysis of the lower extremities and sphincters. A thoracic tumor, by compressing the cord, will produce a spastic paraplegia or an upper motor neuron central type paralysis of the lower extremities, characterized by spasticity, exaggerated muscle stretch reflexes, and pathologic reflexes such as the Babinski sign and clonus. Cervical cord tumors tend to produce quadriplegia or an upper motor neuron paralysis of all four extremities. Establishment of these motor disturbances indicates advanced degrees of cord or root compression. With lesser degrees of compression motor disturbances can be incompletely developed in extent and severity. For example, on gradual compression of the cervical cord by tumor, weakness may gradually develop in the ipsilateral upper extremity and then involve in succession the ipsilateral and contralateral lower extremities and finally the contralateral upper extremity.

SPINAL SUBARACHNOID BLOCK

In spinal cord tumor the cerebrospinal fluid may vary from normal appearance to marked xanthochromia due to increase in total protein content. With massive protein increases some xanthochromic fluids will clot spontaneously (Froin syndrome). Cells may be normal or moderately increased. Occasionally colloidal gold activity is present.

If there is encroachment on the spinal subarachnoid space by tumor above the site of spinal puncture, partial or complete subarachnoid block may be demonstrated by the Queckenstedt test. After free communication between the spinal manometer and the spinal subarachnoid space is demonstrated by rapid rise and fall of manometric pressure on coughing and straining and abdominal compression, jugular compression is carried out. Normally this is followed by prompt rise in manometric pressure coinciding with an increase in intracranial pressure due to impaired (jugular) venous drainage. This pressure increase is transmitted either poorly, or not at all, past a lesion obstructing the sub-

arachnoid channel. It should be emphasized that the Queckenstedt test should not be performed in the presence of blood in the spinal fluid or increased intracranial pressure.

If dynamic block is present, myelography should be carried out immediately. Removal of spinal fluid sometimes shifts spinal cord tumors so as to increase the degree of dynamic block present and to cause rapid exacerbation of symptoms. If this is observed during withdrawal of fluid, reintroduction of cerebrospinal fluid or sterile normal saline solution may decrease the disturbance until laminectomy can be performed.

ROENTGENOGRAPHIC CHANGES

It has been pointed out that alterations in roentgenographic appearance of vertebrae are the rule with secondary spinal cord tumors, that is, those developing within the vertebrae either primarily or after metastasis from distant sites and invading the spinal canal secondarily to produce neurologic symptoms.

Vertebral changes demonstrable roentgenographically may or may not appear with primary spinal cord tumors—those originating from the spinal cord, nerve root, or their soft tissue coverings. Changes when present may consist of scalloping or erosions of the posterior vertebral body surfaces, narrowing of pedicles with widening of interpediculate distances, or enlargement of intervertebral foramina owing to pressure exerted on these structures by the tumor. At times calcific deposits within tumors are seen. Characteristic filling defects are visualized by myelography.

A valuable aid in evaluation of anteroposterior spinal films is a table of normal values for interpediculate measurements.[8] Here the average and extreme upper limits of normal measurements for each vertebral level are shown. Local measurements exceeding these limits should be presumed due to tumor until proved otherwise.

I have thus far considered briefly the commonly encountered varieties of both primary and secondary spinal cord tumors. Now I shall discuss at greater length some of the less frequently seen and described primary spinal cord tumors. Greater familiarity with their roentgenographic and clinical features will promote their more frequent recognition (by roentgenologist, orthopedist, and neurosurgeon).

BLOOD VESSEL TUMORS

Classification.—

Certain vascular tumors cause spinal cord and root compression. Cushing and Bailey[9] in a comprehensive monograph on these tumors, which are usually of congenital origin, classified them as: (1) telangiectases, (2) venous angiomas, (3) arterial angiomas, and (4) hemangioblastomas.

Of these only the hemangioblastomas are true neoplasms.[9] The other three varieties produce clinical symptoms of tumor[10] because of their space-occupying characteristics and potentialities for change and growth, but they have none of the histologic characteristics of neoplasms. Instead, these lesions are angiomatous malformations[11] consisting of hyperplasia, or increase, in the various cellular elements of capillaries, veins, or arteries without qualitative (neoplastic) changes.

Telangiectases. — Thus, the telangiectases are characterized by hyperplastic accumulations of numerous small capillaries lined with endothelium and separated by glia. They most frequently involve the fourth ventricle and pons but also affect other parts of the brain and spinal cord.[11]

Angiomas.—The venous angiomas appear as saccular or convoluted and serpentine varices or as racemose tangles of large, tortuous veins on the surface or within the substance of the brain or spinal cord.[11] Arterial angiomas consist of similar arterial malformations which are quite rare intraspinally.[12] Arteriovenous angiomas are much more common. In these, tangled arterial loops communicate through a capillary bed with dilated and tortuous veins. Netter[7] illustrates one of these lesions in its most common site, intracranially over the junction of the Sylvian and Rolandic fissures.

Frequency and Sites:

Angiomas comprise approximately 2 per cent of both intracranial and intraspinal tumors. The majority are venous, most of the remainder arteriovenous.[5] Very few purely arterial angiomas of the cord have been described. Epstein and his associates[12] found only five cases in the literature and contributed six cases of their own.

Spinal angiomas (hemangiomas) may be: (1) intramedullary, (2) intradural but extramedullary, (3) extradural, or (4) intravertebral. Extradural angiomas generally arise in vertebrae and cause symptoms by extension into the spinal canal with compression of the dura and contained cord and nerve roots. More rarely invasion of the dura and penetration of the cord occur. But intradural and intramedullary angiomas more often arise directly in these locations. Angiomas may remain limited to the body of a vertebra without extension into the spinal canal.[4]

Symptoms:

Vertebral angiomas are usually asymptomatic unless there is extension with encroachment upon the intervertebral foramina or spinal canal.[1] Then signs of radiculitis or slowly advancing cord compression are seen distinguishable from those of extradural or intradural neoplasm only by characteristic changes found in roentgenograms of the affected vertebrae. As more than one vertebra is commonly affected root pains may be indicated at several levels, making the pain more diffuse than that ordinarily observed in extramedullary cord neoplams. At times partial collapse of a vertebra due to extensive angiomatous involvement contributes to the cord compression syndrome.

Intradural and intramedullary angiomas may likewise produce symptoms suggesting neoplasm by compression of the cord and/or irritation of the spinal nerve roots. The signs may be intermittent or progressive, reflecting the capacity of angiomas for growth and change. Hemorrhage, which in minor degree is probably common in intradural and intramedullary angiomas,[5] can also produce neurologic symptoms. Major degrees of hemorrhage within the brain or cord (hematomyelia) or into the subarachnoid space may, of course, be quite devastating.

Diagnosis: Intradural spinal angiomas, unlike the vertebral (and extradural) angiomas, are difficult to diagnose positively before operation. In many cases there may be only a suspicion of the

identity of the lesion.[10] This may, except with arterial angiomas,[12] be aroused at times by the presence of cutaneous angiomas in skin segments corresponding to the level of cord or root involvement. Also angiomas of the face and scalp or retina may suggest the angiomatous and/or hemangioblastomatous nature of intracranial space-occupying lesions.[13] Another curious clue may result from the fact that both angiomas and hemangioblastomas of the cerebellum can be neurogenic causes of polycythemia.[14,15]

Radiographic Findings:

Vertebral and extradural angiomas, which usually arise from the vertebrae, are generally diagnosed by roentgenographic demonstration of characteristic vertebral changes. Bone affected by angioma is usually less dense than normal owing to thinning of trabeculae between the dilated cavernous venous spaces. Sometimes trabeculae are widened, causing increased vertebral density. The greater prominence of trabeculae produced by these changes explains the longitudinally striated appearance of the area of vertebral involvement when seen in longitudinal section or the sponge-like appearance on cross section. Usually the vertebral body is involved, but extension into the pedicles, laminae, and articular processes may occur. Myelographic confirmation of intraspinal extension is often possible.

In intradural and intramedullary angiomas identifying bony changes are not present. Roentgenograms occasionally show calcification in larger angiomatous vessels or in sites of old hemorrhage, but usually nothing distinctive is evident. When seen, erosion of pedicles, laminae, and articular processes is a nonspecific roentgenographic sign of spinal tumor. At times there are congenital malformations of vertebral parts.[4] Subarachnoid block may or may not be present. There may thus be no obstruction to the flow of Pantopaque, or myelography may demonstrate an almost pathognomonic pattern of curved shadows outlining the angiomatous vessel.

Treatment:

Decompressive laminectomy is usually advised to control symptoms of cord and root compression due to spinal angioma. Sometimes more direct surgical attack is feasible. Obliteration of venous angiomas by shrinking the anomalous vessels with very low coagulating currents was first attempted in 1929.[10] Care was necessary to avoid tearing the abnormally thin vessel walls. Since then a series of cerebral and spinal angiomas treated by this and other means with varying degrees of success has been reported.[10] Deep x-ray therapy following decompressive laminectomy and dural incision was considered a useful adjunct in selected cases of venous, arteriovenous, and capillary angiomas.

Probably the first reported successful excision of an intramedullary spinal angioma is that reported in 1948.[16] Extirpation of an arteriovenous angioma in the conus medullaris was undertaken by necessity when a saccular portion of the lesion threatened rupture after exposure.

The experiences of Epstein and his associates[12] suggest that arterial angiomas of the cord are perhaps less amenable to direct treatment than are other types. Their results would indicate that deep x-ray therapy has little effect on the course of the arterial angiomas, in contrast to occasionally beneficial results with venous and arteriovenous angiomas and telangiectases. Radiation was concluded to be inadvisable because of the possibility of further occlusion of small nutrient vessels. Coagulation and ligation or clipping such as advocated in treatment of suitable venous and arteriovenous angiomas[10] were considered disastrous with arterial angiomas.

Hemangioblastomas.—

Hemangioblastomas, which are often associated with angiomas, are to be considered true vascular neoplasms. These tumors are uncommon in the central nervous system, comprising less than 1 per cent of intracranial tumors and 1 to 2 per cent of intraspinal tumors.[5] Hemangioblastomas are usually found within the substance of the brain or cord, often in association with angiomatous malformations, but they may be extramedullary and intradural.

Case Report:

While lifting a refrigerator a 34-year-old male experienced a strain which caused pain to radiate around the left subcostal margin. A few months later a sense of "being cut off" below the waist appeared, and weakness and clumsiness began to occur slowly in the lower extremities. Eighteen months after onset bilateral pyramidal signs were present in the lower extremities, and anal sphincter control was disturbed. Hypesthesia was demonstrated below the eighth thoracic vertebra. Myelograms indicated almost complete block between the fifth and sixth thoracic vertebrae. At operation an almond-sized fleshy tumor was found attached beneath the arachnoid to the dorsal surface of the cord, compressing the latter at the sixth thoracic vertebra. The tumor was attached to the cord by blood vessels coming from the latter. Removal was followed by rapid recovery. Microscopically the tumor consisted of a spongy mass of capillaries and pseudoxanthomatous cells. At one pole a large cyst lined with endothelial cells comprised a considerable portion of the tumor.

Diagnosis: Hemangioblastoma, intradural, extramedullary.

The hemangioblastomas appear to result from congenital inclusion of blood-forming mesenchymal elements within the dorsal midline of the central nervous system during the process of invagination and isolation of the neural tube.[5] These tumors contain capillaries lined with large endothelial cells of various shapes. In some tumors these endothelial cells form solid masses or separate into bands forming sinusoid cavernous spaces. Between capillaries, fat-laden pseudoxanthomatous cells of foamy appearance may be seen. On the basis of characteristic differences in cellular and vascular arrangements, three general types of hemangioblastomas are recognized: (1) predominately capillary, (2) predominately cellular, and (3) predominately cavernous.

Craig and Horrax[13] illustrate the appearance at operation of an intramedullary spinal hemangioblastoma. The occurrence of similar lesions in the cerebellar hemispheres of a sister and the mother of the patient typifies the familial character of these neoplasms as well as their greater incidence in the cerebellum than in the cord.

Hemangioblastomas frequently become cystic through active transudation of fluid from the vessels in the tumor. Distention of the tumor and compression of the surrounding brain or cord tissue result which may give rise to cysts within the cord, resembling syringomyelia, or cysts within the brain, usually the cerebellum.

EPIDERMOID AND DERMOID TUMORS

An interesting group of clincally important intraspinal lesions originating with embryonic accidents is composed of the epidermoid and dermoid tumors with teratoids and teratomas of the spinal canal and communicating dermal sinuses. The clinical picture of these tumors is characterized by long standing symptoms of spinal cord or nerve root compression which may date from childhood. Males are more often affected, and an intermittently draining midline sinus is sometimes seen in the lumbosacral region. The benign character and slow rate of growth of the dermoids and epidermoids promote satisfactory relief by operative removal.

Sachs and Horax[17] contributed two cases of intraspinal dermoids and an exhaustive review of the literature. An additional case reported by Moore and Walker[18] illustrates the important features of orthopedic and radiologic interest of this group of tumors.

Etiology.—Dermoid tumors are caused by congenital disturbances during formation of the neural tube which result in inclusion of dermal elements in the latter. The ectopic stratified squamous epithelium undergoes keratinization and desquamation, giving rise to the dermoid cyst.[6] Epidermoids are of similar origin and differ from the dermoids chiefly in containing hair. Teratoids and teratomas are cystic or solid tumors containing mixtures of ectodermal, mesodermal, and, more rarely, entodermal elements. The less complex and bidermal examples are often designated as teratoids.[5] They appear to result from differentiation of ectopic embryonic cell rests of polydermal potentials.

SUMMARY

The primary and secondary spinal cord tumors have been classified and the commoner varieties described. Mechanisms of symptom production and principles of diagnosis have been discussed. Although less common, the blood vessel tumors and congenital inclusion tumors affecting the spinal cord and roots have also been described.

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Nutritional and Other Disturbances of Bone Involving Primarily the Endochondral Ossification Centers of Growing Long Bones

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It is the purpose of this presentation to discuss the underlying pathologic physiology responsible for the changes observed roentgenographically in the bones of an individual suffering from faulty or disturbed bone nutrition.

It is realized that such changes occur throughout the entire osseous system; however, our discussion will be centered around the changes occurring in the region of the epiphysis. In order to understand and fully appreciate these changes, it is necessary to review the histology of the normal growing epiphysis. The epiphysial and diaphysial areas remain separated by uncalcified cartilage called the epiphysial disk or plate. When bone maturation is achieved at approximately 20 years of age, these disks or plates disappear. These plates should be of utmost concern to roentgenologists considering the various pathologic conditions which involve the growing bones of children. A detailed study will therefore be made of this area.

Histologically, five zones are demonstrated and may be described as follows:

The first zone lies nearest the secondary center of ossification, the epiphysis, while the fifth zone lies nearest the primary center of ossification, the diaphysis. The first zone is made up of resting cartilage cells awaiting their turn to take part in bone growth.

Zone two consists of proliferating cartilage cells which are normally arranged in columns. The cells at this level are actively dividing. This is a zone of cartilage cell maturation. These cells are large and tend to increase the width of the epiphysial plate.

Zone three contains cells in various stages of maturation.

Zone four consists of degenerated cartilage cells which are dying and which become surrounded and invested with a calcified matrix, providing there is sufficient calcium and phosphorus available in the plasma and tissue fluids. This zone of provisional calcification forms a bridge and gives rigidity to the gap between the hyaline cartilage and spongy bone.

This layer of spongy bone constitutes the fifth and final zone, namely, the zone of proliferating osteoid. This fifth zone consists of embryonic connective tissue, or primitive bone marrow, which is made up of mesenchymal cells and blood vessels. The end points of bone formation take place in these last two zones. Loops of blood vessels and osteoclasts from the zone of proliferating osteoid penetrate the calcified matrix in the zone of provisional calcification and open up the cartilage cell capsules.

In this way, vascular embryonic connective tissue is laid down in communicating canals whose walls are formed by cartilage cells. The portion of embryonic marrow nearest the cartilage matrix turns into a layer of osteoblasts. Between the osteoblasts and the cartilage a thin new layer appears which thickens and surrounds the contour of the adjacent bars of cartilage. This new layer usually calcifies as it is deposited, forming osseous tissue or bone.

If calcification is not prompt, owing to local inadequate supply of bone minerals, osteoid tissue appears in excess, and the condition known as rickets is present. From the foregoing outline, it can readily be seen that three basic processes are continually taking place within the epiphysial disk:

1. Cartilage cell development and maturation
2. Calcification, degeneration, and death of cartilage cells
3. Interaction between calcified dead cartilage cells and embryonic connective tissue which forms calcified osseous tissue or new bone.

Osteoclastic and osteoblastic activity is therefore quite vigorous in the area immediately adjacent to the diaphysial side of the epiphysial disk. It is in this area that endochondral ossification takes place.

As pointed out at the beginning of this discussion, it is our hope to relate the normal and pathologic histology of the epiphysial plate and adjacent structures to known roentgenologic changes that identify certain diseases. Four major conditions have been selected which show marked changes in the region under discussion, namely, scurvy, rickets, hypervitaminosis D, and congenital lues, as well as certain other pathologic bone conditions considered important from the standpoint of differential diagnosis.

SCURVY

In scurvy, attention is called to the zone of provisional calcification and the zone of proliferating osteoid. With insufficient amounts of vitamin C in the diet, bone formation is brought to a standstill, yet the normal process of bone absorption still goes on, and the bones become rarefied and fragile. Vitamin C specifically causes cessation of cartilage and bone matrix formation. Without this collagenous matrix, osteoblastic proliferation all but ceases, and cartilaginous growth, although still possible, results in the formation of imperfect cartilage. Although cartilage formation is imperfect and reduced, the amount of cartilage matrix substance undergoing calcification is abnormally great. Since osteoblastic activity practically ceases, little or none of the calcified cartilage in the zone of provisional calcification is converted into bone. The resulting broad, dense epiphysial line of calcified cartilage is recognized radiographically as Fraenkels' white line or the Trummerfeldzone.[1]

A dense ring at the periphery of the secondary epiphysial centers, giving rise to the appearance of an ossification center within an ossification center, is similar to the broadened zone of provisional calcification just mentioned as occurring at the diaphysial ends of long bones and is produced by the same pathologic factors.

An atrophic poorly defined zone of osteoid tissue is visualized roentgenographically on the diaphysial side of the epiphysial line. Osteoblasts are few in number, and their activity is reduced; therefore, a transverse band of radiolucency is produced which is referred to as the scurvy zone, or the framework marrow zone.[2]

There is no impairment of calcification in scurvy such as in rickets. The problem is an impairment in osteoblastic activity which results in the changes described.

RICKETS

Rickets is a disturbance of calcium metabolism, not of bone growth. This condition develops secondary to vitamin D deficiency, and attention is again drawn to the zone of provisional calcification and the zone of proliferating osteoid. Since vitamin D is not absorbed and utilized, the serum calcium level is maintained at the expense of other body tissues, especially bone. It is therefore easy to understand the reason that calcium is not laid down in the intercellular matrix of the zone of provisional calcification. Because of this fact, complete disordered proliferation of cartilage continues, as projecting tongues, into the marrow spaces, with corresponding long and short projections of uncalcified osteoid from the zone of proliferating osteoid extending into the cartilage zone.

The wide epiphysial line visualized radiographically is due, therefore, to unhampered production of cartilage which does not calcify, coupled with the excessive deposition of uncalcified osteoid emanating from the zone of proliferating osteoid.

Since this continually forming osteoid fails to calcify, the metaphysial ends of long bones enlarge. This change is illustrated at the anterior ends of the ribs and is referred to as the rachitic rosary. A rather poor quality of endochondral bone may be formed at the periphery of the metaphysis and end completely in the center, a condition which produces cupping and fraying at the ends of long bones and is referred to as the wine glass deformity of rickets.

HYPERVITAMINOSIS D

In hypervitaminosis D, a reversal of the changes characteristic of rickets is visualized in the zone of provisional calcification and the zone of proliferating osteoid.

The zone of provisional calcification becomes widened and is exceedingly dense. Abnormal fixation of calcium is present in the zone of proliferating osteoid; therefore, this region is unusually dense. Alternating periods of supernormal and normal intake of vitamin D result in formation in the metaphysis of alternating transverse bands of calcific density and normal density; the width of these bands depends upon

the duration of normal and/or excessive intake of vitamin D. It is only with the transformation of primary into secondary spongiosa that the structural relationships become normal.

CONGENITAL SYPHILIS

Congenital syphilis is blood borne to the fetus via the placenta. Early destructive changes are created in the region of the metaphysis, since the spirochete tends to localize in highly vascular regions.[3]

In congenital syphilis, two pathologic processes are in operation; the proliferative cartilage is poorly formed but has not lost its power to produce an intercellular matrix which calcifies readily. The more sensitive osteoblasts, on the other hand, which normally convert this cartilage matrix into bone, are more seriously damaged. They are unable to perform this function, and, in consequence, calcified cartilage matrix accumulates in excessive quantities at the epiphysial line, forming a dense lattice which is easily seen in the roentgenogram.

In syphilis, unlike in scurvy, bone growth may be inhibited temporarily. Periods of quiescence alternate with periods of growth. As a result, the zone of provisional calcification may partially resorb, and thus two or more transverse lines of calcification may become visible on the roentgenogram.

The transverse band of decreased radiodensity visualized proximal to the zone of provisional calcification is formed by the presence of syphilitic granulation tissue in the zone of proliferating osteoid. Syphilitic granulation tissue may cause bilateral bone destruction, affecting the proximal and medial surfaces of the shafts of the tibias near the metaphyses. When this zone of radiolucency is visualized, it is considered pathognomic of lues and is referred to as Wimberger's sign.

TRANSVERSE LINES OF INCREASED DENSITY

This presentation would not be complete without pointing out the fact that transverse lines of increased density in terminal segments of growing bone may be found in apparently healthy as well as in chronically ill infants and children.[3] These lines are not necessarily present when bone growth is retarded for a period; on the contrary, they may be the result of excessive bone growth.

Lead, bismuth, radium, phosphorus, and fluorine may be responsible for increased bone density traversing the metaphysial ends of growing bone. Some authorities[3,4] feel this increased density visualized roentgenographically is due to excessive calcification of the cartilage matrix in the zone of provisional calcification. Others[1,5] hold that the increased density results primarily from actual deposit of these various substances in the highly vascular metaphysial portions of bone.

Suffice it to conclude that such toxic changes affect primarily the zone of provisional calcification and/or the zone of proliferating osteoid, according to the etiologic chemical element involved. Excess calcification is present involving the cartilage matrix in the former zone, and/or associated stimulation or depression affecting the embryonic layer of the latter zone results.

DISCUSSION

The underlying pathologic conditions responsible for certain roentgenographic changes have been discussed as they affect the epiphysial ends of the long bones of growing children suffering from scurvy, rickets, hypervitaminosis D, and congenital syphilis.

It has been pointed out that the zone of provisional calcification shows considerable thickening and increased density in scurvy, syphilis, and hypervitaminosis D and disappears in rickets. Scurvy, rickets, and syphilis present bands of radiolucency crossing the metaphysial ends of long bones. The bands in these conditions vary in width and configuration, depending upon the condition producing them, whereas hypervitaminosis D produces transverse bands of normal bone density between transverse zones of increased density. This fact, coupled with the lack of other findings, roentgenographically differentiates hypervitaminosis D from scurvy, rickets, and syphilis.

In scurvy, the band of lessened density is very narrow and is seen lying on the diaphysial side of the white line of Fraenkel or the zone of provisional calcification. This zone occurs largely because fibroblasts have replaced osteoblasts in the zone of proliferating osteoid.

In rickets, cartilage cells proliferate in disorderly fashion, while the surrounding connective tissue matrix fails to calcify. The development of osteoid is normal, but, here again, calcification is imperfect. These facts account for the rachitic intermediate zone, or rachitic metaphysis. Deposition of calcium in the zone of provisional calcification and the zone of proliferating osteoid has failed because lack of sufficient vitamin D intake has resulted in maintenance of the blood calcium level at the expense of bone mineralization. Failure to visualize the epiphysial plate roentgenographically is therefore helpful in determining the presence of rickets. Resulting absence of the zone of provisional calcification (the epiphysial plate) in rickets differentiates this condition from scurvy and syphilis.

In congenital lues, the zone of proliferating osteoid is the site of major involvement and bears the brunt of the syphilitic metaphysitis. This zone shows marked widening and irregularity similar to rickets, but the zone of provisional calcification, while absent in rickets, is roentgenographically more prominent than ever in congenital syphilis.

Certain toxic changes affecting the metaphysial ends of growing bone have been mentioned. Their differential diagnosis from scurvy, rickets, hypervitaminosis D, and congenital syphilis is based primarily on clinical laboratory evaluation rather than roentgenographic evaluation.

SUMMARY AND CONCLUSIONS

We have attempted to review the normal histologic and pathologic findings of the epiphysial plate and adjacent metaphyses and to correlate these zones with the roentgenographic findings in scurvy, rickets, hypervitaminosis D, and congenital lues. Thus the zones of provisional calcification and proliferating osteoid in the epiphysial plate are of utmost significance to the roentgenologist in the study of nutritional deficiencies.

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Bronchologic Aid to the Radiologist

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In pulmonary conditions, the internist tapping, looking, listening on the outside, the roentgenologist, in a sense, looking through the patient, and the bronchoscopist looking inside the tracheobronchial tree and obtaining specimens of tissue and uncontaminated secretions for the laboratory form a diagnostic team which never has been equaled.[1]

The patient has not been treated fairly if diagnostic bronchoscopy is omitted in any case in which there are physical or roentgenographic signs of bronchial obstruction, such as unexplained cough or hemoptysis or a wheeze.[1] Signs of obstructive emphysema or obstructive atelectasis are also definite indications for diagnostic bronchoscopy.

Bronchoscopy is also indicated in different clinical entities such as pulmonary abscess, bronchiectasis, emphysema, asthma, obstructive atelectasis, tuberculosis, and benign and malignant tumors.[1]

PULMONARY ABSCESS

It is a mistake to regard bronchoscopy and surgery as competitive or opposed methods of treatment. They should be thought of together, as bronchoscopy and surgery versus disease rather than as bronchoscopy versus surgery. Bronchoscopy constitutes an integral part of both the medical and surgical, or the conservative and radical, treatment of pulmonary abscess.

Sometimes bronchial obstruction of one kind or another, a foreign body, tumor, or inflammatory stenosis, is present in pulmonary abscess, and in these cases it is extremely important to discover and eliminate the obstruction. Routine diagnostic bronchoscopy is, therefore, indicated in pulmonary abscess.[1-3]

Late cases[4,5] of pulmonary abscess may be defined as those which have a large or poorly drained cavity or an atelectatic or fibrous reaction around the wall of the cavity. Late cases are definitely in need of surgical therapy. A late case is also one which may be complicated by daughter cavities even though in terms of time they may be found within a few weeks from the onset. Nye[6] offers one other indication for surgical management of lung abscess: when the abscess remains unchanged on the roentgenogram for 2 to 3 weeks, even if there has been amelioration of symptoms.

A viewpoint still held by some[5,7] that the lower lobe is the commonest site for abscess formation seems to me to require modification. It has been my experience that the common site for abscess formation is the upper lobe. In fact, of ten cases which I have seen this past year, the lower lobe (right) has been invaded only twice. Another disappointing finding, radiographically, is that only 50 per cent of lung abscesses show convincing evidence of cavitation. Incidentally, 25 per cent of all lung abscesses are secondary to bronchial carcinoma of the periphery.[8]

BRONCHIECTASIS

It must be understood first that bronchiectasis ordinarily cannot be accurately diagnosed without the use of a contrast medium such as Lipiodol. In a few cases, reported by Jackson and Bonnier,[10] apparent bronchiectasis has proved to be "reversible" and has disappeared after a series of bronchoscopic treatments in all cases of "colds which do not clear up," "unresolved pneumonias," and similar conditions.

In chronic, well established bronchiectasis with profuse foul sputum and hemotysis of varying severity, bronchoscopic treatment can be of considerable palliative value, but the only hope of cure is lobectomy or pneumonectomy. Preoperative and postoperative bronchoscopy is of great value to the surgeon in these cases. Bronchoscopic aspiration should also be performed when operation is contraindicated by the extent of the disease or by the patient's general condition.

Bronchiectasis is a segmental disease,[3,9] and it may be present in any one of the 18 segments of the two lungs. In about 35 to 50 per cent of cases it occurs bilaterally. Bronchial obstruction, partial or complete, is responsible for many of the clinical manifestations of bronchial asthma. A reduction in the lumen impairs ventilation and gaseous interchange, which, in turn, is followed by changes in the circulatory dynamics. The bronchus may be obstructed by spasm, fibrostenosis, a foreign body, or lymph nodes which may compress, encircle, or perforate the bronchus. Often these factors combine to produce bronchiectasis. Bronchial segments are affected most often, and they may be completely or partially obstructed, with resultant atelectasis or emphysema. With structural changes in one or more segments, there is an increase in the negative intrapleural pressure, with a greater dilatation effect on adjacent segments. The altered mechanics make it more difficult for the patient to raise secretions.

Bronchoscopy is performed for diagnostic and therapeutic reasons.[3] It may reveal the source of purulent sputum or pulmonary bleeding and at times may impress the observer with the significance of the presence of minimal bronchiectasis.

OBSTRUCTIVE ATELECTASIS

The first form of obstructive atelectasis to be recognized other than that of foreign body origin is the postoperative variety. It is found that the commonest postoperative pulmonary complication is not pneumonia but atelectasis, easily curable by bronchoscopic aspiration. Many intrabronchial lesions, whether inflammatory or neoplastic in character, may also produce atelectasis.

Diagnosis of a tumor arising in a segmental bronchus is usually made early although with somewhat more difficulty than in cases where the larger bronchi are involved. A lesion arising in a segmental bronchus[11] will obstruct the segmental bronchus almost at the start. For this reason, symptoms frequently have their onset in a more or less acute febrile illness, the result of segmental obstruction and secondary infection. An x-ray film made at this time will show changes limited to a single bronchopulmonary segment.[12] There will be a wedge-shaped area of density

extending peripherally from the hilus, and the involved segment can usually be identified in the lateral view.

LUNG TUMOR

Impairment by an obstruction of the normal mechanism of the bronchial drainage by a lung tumor results in secondary infection.[1,2,11] This occurs in one third to half of the cases and may at first resemble one of the common acute and recurrent lower respiratory infections. At this stage of the disease the patient is often treated for various types of pneumonia, bronchitis, influenza, or pleurisy. Recurring pulmonary infection, frequently and mistakenly called virus pneumonia, should awaken any suspicion not previously aroused. Unresolved pneumonia, which is an erroneous term, is really the manifestation of a lung abscess, bronchogenic carcinoma, bronchiectasis, or tuberculosis.[11]

As the degree of the bronchial obstruction progresses[13] there is an interval when the check-valve mechanism gives rise to signs of obstructive emphysema. This period usually is a transitory one, and in those cases of obstruction which become complete, the signs of emphysema are soon succeeded by those of atelectasis.

Rigler and his associates,[14] in studying the duration of carcinoma of the lung in 127 cases, were satisfied that lung emphysema, whether segmental, lobar, or unilateral, is one of the most important early roentgen signs. Therefore, it is imperative that not only inspirational but expirational films be taken to substantiate or negate this early radiographic finding of lung tumor. Their most important contribution to the early recognition of chest tumors is the realization of the presence of unilateral hilar enlargement. On the basis of the survey of 127 cases, it was found that nine out of ten cases in which the transverse diameter of the hilus measured more than 13 cm. will be abnormal, that is, probably bronchogenic carcinoma is present. Nine out of ten cases in which one hilus is 1.7 cm. greater than the opposite hilus will also be abnormal.

Only 30 to 35 per cent of lung neoplasms are actually resectable,[15,16] and half of these resections are classified as palliative rather than curative because of the extent of the lesion. Therefore, it is imperative that early diagnosis and evaluation of a lung tumor be made.

Irrespective of whether clinical symptoms are present, bronchoscopy should be performed[11] not only with the expectancy of obtaining tissue for biopsy but with the intention of ruling out involvement of the main bronchus in its proximal portion. Further examination should evaluate any indirect evidence of mediastinal involvement such as compression or fixation of the lower trachea or main bronchus, widening or fixation of the carina, and/or paralysis of one vocal cord. A negative finding in bronchoscopy, biopsy, or in a test of bronchial secretions does not rule out carcinoma. In other words, a positive finding is of great significance but a negative finding does not rule out the possibility of carcinoma. In some suspicious cases repeated bronchoscopic examinations are indicated in an attempt to obtain cytologic confirmation.

I agree with those who feel that the contraindications to resection of lung tumors are actually more important to the surgeon than the indications.[5,6,11] These contraindications are usually not verified radiographically. They are paralysis of a vocal cord, particularly of the left, any disturbance in the contour of the trachea, widening of the angle of bifurcation of the trachea, interference with free movement of the carina, encroachment on any of the bronchial subdivisions, deformity, fixity, or rigidity of the main bronchi, and evidence of metastasis not only to bones but also to remote organs and pleural fluid.

TUBERCULOSIS

Until a few years ago, bronchoscopy was thought to be contraindicated in tuberculosis. The bronchial lesions of tuberculosis are chiefly either flat ulcerations or ulcerogranulomas.[17,18] The presence of a tuberculous bronchial lesion associated with nonsuppuration should be regarded as a contraindication to collapse therapy and also to a pulmonary resection.

Bronchoscopy should be performed when roentgen studies do not reveal evidence of tuberculosis, but sputum examinations continue to show the presence of Mycobacterium tuberculosis.[3] It is also valuable in unexplained pulmonary infections when sputum is not available for examination. With bronchoscopy, secretions can be obtained for study, and those obtained from below a stenosed bronchus may contain the bacillus of tuberculosis whereas secretions obtained from above the point of obstruction may not.

ASTHMA

A most interesting and valuble use for bronchoscopy is in cases in which chronic bronchitis or asthma produces attacks of chills and fever.[19] Most frequently the attacks of fever will last from 3 to 5 days, and the patient will be relieved by coughing up a small amount of purulent material. Bronchoscopic examination may reveal narrowing of a bronchus, and dilatation of the bronchus may liberate a small amount of retained secretion. After bronchoscopy, marked improvement often will occur in the respiratory symptoms and the attacks of fever will disappear. The bronchi that are most frequently involved in this process are the superior divisions of the lower lobe bronchi.

CONCLUSION

It must be kept in mind that many different types of pathologic conditions in various locations in the lungs and mediastinum have similar x-ray manifestations,[2,20,21] and a definite diagnosis cannot always be established even after the employment of all methods of thoracic diagnosis such as bronchoscopy, bronchography, esophagoscopy, examination of sputum, and cytologic examination of bronchial secretions.

Faced with a too frequently questionable diagnosis and questioned by those who demand absolute proof of the presence of the lesion, the physician must ask himself, "Will my patient pay with his life if I advise a wait-and-see policy?"

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Fungous Diseases of the Chest

Their Differentiation

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Recognition and differentiation of pulmonary mycotic infections from other pathologic processes involving the lung fields are often of vital importance in the proper management of the case.

The exact diagnosis of these lesions is often difficult radiographically for they may simulate other entities. Many times serologic procedures, cutaneous tests, and smears and cultures from surgically removed lesions, sputums, and draining exudates are of inestimable aid.

The purpose of this paper is to attempt to erect some roentgenographic signposts for differentiation.

It is well to recall that, though there are over 100,000 species of fungi, relatively few are pathogenic for man, and still fewer are capable of producing pulmonary mycotic disease. Those that show pathogenicity for man include Actinomyces, Blastomyces, Coccidioides, Monilia, Torula, Aspergillus, Histoplasma, Toxoplasma, Streptothrix, Leptothrix, Sporothrix, Penicillium, and Mucor.[1,2] Some of these are quite rare. I shall discuss only those considered most important clinically.

All of these disease processes have certain characteristics in common. In the acute phase they resemble clinically any other type of infection. In the chronic phase they resemble tuberculosis because they react in pulmonary tissue as a granulomatous inflammation.[3] Since they are fundamentally alike anatomically it follows that they produce shadows on the roentgenogram of marked similarity to one another and to tuberculosis, whether nodular or cavitary.[4] Being granulomatous there is an exudative reaction (fluid elements of the circulation) and a productive reaction (the reticuloendothelial or wandering cells).

The deposit of granules in the area leads, obviously, to the term "granuloma." In the presence of certain injurious agents a necrotizing reaction may result, with breakdown of the lesion and a productive reaction.

Mycotic infection is but one of the etiologic agents producing such a reaction, for viral, bacterial, chemical, and physical agents may also produce granulomas, Boeck's sarcoid being an example of a granuloma of unknown etiology. With this knowledge of their basic histopathology it is easy to see that they may not only look much alike, but that they have a tendency to form discrete or confluent tubercles or nodules which may become necrotic and abscess in some cases, the intervening lung tissue being hyperemic and the seat of bronchopneumonic consolidations.

As a group, these mycotic infections usually seem to be distributed midzonally or basally on the roentgenogram, with prominent hilar shadows and frequently pleural involvement, but the exceptions to this are enough to merit a more detailed study of the entities.

COCCIDIOIDOMYCOSIS

This mycotic infection, once almost solely seen in the southwestern United States, is now widespread in this country.[2] It must be an early consideration in any respiratory infection where the disease is endemic. A negative intradermal test with 0.1 cc. of 1:100 coccidioidin rules out the disease in all but the most severe disseminated infections. A positive precipitin or complement fixation test is found only in an active infection, though some mild cases may give a false negative reaction. Final proof is demonstration of Coccidiodes immitis in a culture of sputum and confirmation with guinea pig inoculation.

On the roentgenogram the early pneumonic phase of coccidioidomycosis may so resemble primary atypical pneumonia or rheumatic pneumonitis that only the laboratory can differentiate them, for the infiltrates will vary from a veil-like haze to consolidations approaching those of lobar pneumonia.[5] At this stage the very high incidence of chest pain, often simulating coronary occlusion or fractured rib, may help to differentiate it from the more common respiratory infections. The presence of a spiking fever, with temperatures from 100 to 102 F., leukocyte count from 10,000 to 12,000, and 3 to 15 per cent eosinophilia may be of further aid in reaching a diagnosis. If in 1 to 2 weeks painful nodules develop on the shins or elsewhere (erythema nodosum) or skin lesions of erythema multiforme are found, diagnosis becomes easier.

Serial chest films show the hilar type infiltrate resolving rapidly, usually in 1 to 2 weeks. The very soft parenchymal infiltrates usually disappear in 2 to 4 weeks. The denser consolidations clear slowly and give rise to the so-called persistent coccidioidal infections which are worthy of classification:[6]

1. Nodular Parenchymal Foci: These are often discovered on routine chest films though there may have been a few chest pains, weakness, and so forth. Often there is a history of an acute febrile illness 2 to 10 months previously. On the average the elapsed time

between the occurrence of a zone of pneumonitis and nodule formation is about 5 to 6 weeks. These nodules average about 2 cm. in diameter and are strikingly similar to solitary or bilaterally disseminated metastases or uncalcified primary tuberculosis foci. Though they may disappear or diminish in size, many do not change for long periods or may go on to eventual calcification.

2. Coccidioidal Cavities: Surprisingly these may be discovered without prior clinical evidence of the disease, but often are found on investigation because of chest pain, fatigue, or hemoptysis. They can develop either from a zone of pneumonitis or by central excavation of nodular foci. As a rule, the patient with a prolonged severe acute illness is more prone to develop cavities than nodules. Oddly enough, clinical signs of activity of the disease are often absent even in the presence of fluctuations in the size of the cavity. These cavities are very slow and indolent in their evolution. Over many months they may collapse only to reappear suddenly; they melt away; they may end as small nodules; they may trap air and look like congenital cysts; they may contain fluid with a surrounding infiltrate and simulate lung abscesses or ulcerative tuberculosis.

3. Persistent Coccidioidal Pneumonitis: This type of roentgen finding is usually seen in a severely ill patient with a very high fever and high sedimentation rate. The infiltrate may involve one-half to one-third of a lung field. These cases do not focalize as nodules or cavities in the usual 2 to 6 weeks' period required for stabilization, but rather may require as long as 6 to 21 months to clear, and there may be a course of remissions and exacerbations.

4. Mediastinal and Hilar Adenopathy: These may readily be confused with Hodgkin's disease, primary tuberculosis, Boeck's sarcoid, or even bronchogenic cancer, especially since they occur in persistent types of coccidioidal infections, with a severe clinical course often proving fatal. The sedimentation rate remains high as long as adenopathy is present, which may be for months.

5. Pleural Effusion: This may be expected in about 20 per cent of cases, varying from a very small amount to massive, persistent effusion lasting for months.

6. Fatal Disseminated Coccidioidomycosis: At the onset these cases may resemble acute pneumonic cases which resolve in 2 to 6 weeks, but the radiologist should beware of the case in which the temperature fails to fall; in which complement fixation titer, instead of decreasing, rises as high as 1:64 or 1:128, or in which the sedimentation rate persists at 50 to 100 mm. per hour. Though the film is dominated by hilar and mediastinal adenopathy, there is often a miliary "seeding," more rapid, less uniform, and more confluent than in tuberculosis. Within a week or two of the miliary spread, small punched-out osteolytic areas may be found in the ribs, scapulae, or clavicles, quite similar to metastatic cancer in appearance. At autopsy practically every organ in the body is found involved by the coccidioidal granulomas.

It cannot be emphasized too strongly that coccidioidomycosis, like syphilis, is truly, among the mycotic infections, the "great imitator."

ACTINOMYCOSIS

Metastatic pulmonary lesions often result from hematogenous spread of the infection from the face or intestinal tract. At first these lung lesions may be indistinguishable from lobar pneumonia or lung abscess, though occasionally they may resemble bronchopneumonia, tuberculosis, or silicosis with infection. As the disease progresses there is a marked tendency to chest wall or pleural involvement with the formation of a granuloma, abscess, or osteolytic process involving the ribs.[1] The typical ray fungus of actinomycosis is usually found even when the culture of the abscess wall is negative. Rarely the disease may produce a miliary spread not unlike that of tuberculosis.

BLASTOMYCOSIS

Like actinomycosis this process is usually found in farmers and cattlemen and is the result of inhalation of the spores of Blastomyces dermatitidis, a large double contoured parasite. These are influenza-like symptoms, and positive identification is made from material obtained from skin lesions, from sputum examination, or from complement fixation reaction.

Radiographically a rather characteristic finding is a dense, irregular shadow extending from the hilus to the lung periphery.[2] It may, however, like actinomycosis, resemble tuberculosis, even to the point of miliary spread.[1]

ASPERGILLOSIS

Like actinomycosis and blastomycosis, this disease is found among farmers, grain handlers, and those coming in contact with pigeons and parrots. The x-ray film reveals countless widespread, uniform, small, soft, rounded infiltrations throughout both lung fields.[3] The lesions are larger than those of miliary tuberculosis, but the patient is not profoundly ill as in that disease. It bears a resemblance to a second-stage silicosis with multiple silicotic nodules. It heals with multiple calcifications of unequal size, hence it must be a consideration in any case ending in widespread calcific distributions. It must also be considered when hemoptysis occurs, for this symptom may be associated with relapses of aspergillosis.

SPOROTRICHOSIS

This is still another disease in which the offending organism, the Streptothrix, is found among farm laborers and nursery workers. Radiographically there are areas of consolidation within which necrotic zones develop. This entity, like actinomycosis, aspergillosis, and moniliasis, may resemble nodular silicosis or result in pleurocutaneous fistulas.[7]

MONILIASIS

It has seemed to me that with the widespread use of antibiotic therapy there has been an increasing number of cases in which Monilia albicans is rampant in the sputum, at times with apparently complete suppression of all other pulmonary flora. Since Monilia is often present in the upper respiratory tract of healthy individuals, its presence is not pathognomonic of the disease when found in the sputum, and many previously reported cases of pulmonary moniliasis are open to question.[2] The diagnosis, then, of true moniliasis must rest on the exclusion of other lung disease, for the presence of Monilia may be only a "red herring" obscuring the true pulmonary disease. In well-authenticated cases there are increased perivascular markings in the lower lobes, often progressing to irregular infiltrative areas which either become nodular like those of silicosis or resemble pyogenic bacterial infection.[7]

TORULOSIS (CRYPTOCOCCOSIS)

This is a rare but interesting entity, encountered four times in 537,135 admissions at New Orleans Charity Hospital in the last 10 years.[8] Despite its

rarity several authors[8-10] have felt it to be of sufficient importance to describe it rather thoroughly.

It is caused by the Torula histolytica, which, though found at times on normal skin, in the feces, and in the female genital tract, may produce destructive granulomatous lesions in the lungs and nervous system. On the x-ray film the pulmonary lesions may vary from triangular to round shape, or they may appear as cotton-like areas of basal infiltration. The fungus may be absent in the sputum or overlooked by the inexperienced technician. It may be found only in the spinal fluid. Often the roentgenologist will suspect tuberculosis,[9] but he should bear in mind the possibility of torulosis when he sees the following: a parenchymal pulmonary lesion which is relatively asymptomatic, has inflammatory characteristics, changes little with time, and shows no other evidence of neoplasm.[8] Later, the onset of central nervous system symptoms or the finding of submetaphyseal decalcifications may clinch the diagnosis.[9] This infection is important to the pediatrician and the obstetrician for it may be acquired in utero, during delivery, or early in the neonatal period, even proving fatal in a few weeks in some cases.[10]

HISTOPLASMOSIS

Though it is common knowledge that the pathogenic fungus Histoplasma capsulatum may be the cause of clinically unimportant calcifications in the lungs and mediastinal nodes, it is well to remember that it can be a serious and frequently is a fatal disease involving almost all the viscera.[11]

An antemortem diagnosis of histoplasmosis, based solely on roentgen findings, is rarely possible, for a more diversified group of pulmonary changes could scarcely be found anywhere. The most consistent finding has been a widespread granular type lesion throughout both lungs, which unfortunately is usually terminal and readily confused with miliary tuberculosis, pneumoconiosis, sarcoidosis, lymphoblastoma, bronchopneumonia, lymphogenous pulmonary metastases, and various fungous diseases. On occasion the fungus may be found in the sputum, or a nonfatal case may show hilar and mediastinal adenopathy, or a very indolent, slowly resolving parenchymal lesion, as in certain cases of coccidioidomycosis, may be encountered.

TOXOPLASMOSIS

This disease process, involving the central nervous system in the manner of torulosis and causing intracranial calcifications as in torulosis, histoplasmosis, and blastomycosis,[12] is caused by a yeastlike parasite (Toxoplasma) which affects rabbits, squirrels, dogs, and birds, and has been found to cause disease in man. Clinically it resembles typhus and Rocky Mountain spotted fever. Radiographically it appears as diffuse, interstitial pneumonitis with shotlike areas of induration in the lung fields which measure from 1 to 4 mm. in diameter, simulating virus pneumonia.[2]

BOECK'S SARCOID

From the standpoint of differential diagnosis it might be well to mention briefly Boeck's sarcoid, a nonspecific granuloma of which much has been written,[13] for this process may occur as a miliary distribution simulating any one of the miliary-type entities or as a nodular process resembling any one of the fungous lung diseases. About two-thirds of sarcoid cases show bilateral hilar plus right paratracheal adenopathy, often in an apparently healthy person. This finding, plus biopsy of a node, the finding of sarcoid bone lesions, the presence of peripheral painless adenopathy, and/or cutaneous and ocular lesions, may aid in differentiating Boeck's sarcoid from mycotic lung infections. From the laboratory a report of hyperglobulinemia with reversal of the albumin-globulin ratio, eosinophilia, increased sedimentation rate and elevated serum calcium and phosphatase values all point toward a possible sarcoidosis.

CONSULTATION

It is well to remember that whenever we, as radiologists, encounter a diffuse granulomatous process of both lungs or of the mediastinal lymph nodes in which diagnosis is in doubt, the surgeon may perform the simple procedure of a scalene node biopsy, even when the node is not palpable. This will frequently prove of great help in arriving at the proper diagnosis.[4] Furthermore, the surgeon should be consulted whenever the diagnosis of any granulomatous, pulmonary mycotic lesion in inconclusive; when there are pulmonary cavities, mycotic or nonmycotic; when a granuloma is mimicking a pulmonary neoplasm; when hemoptysis is associated with a mycotic infection, or when there is infection or spontaneous rupture of a cavity.

This is especially true today since conservative pulmonary excision may remove the major focus of the disease with the minimum sacrifice of functioning pulmonary parenchyma. Furthermore, it may not only be the sole way of arriving at a positive diagnosis; it may save the patient's life.

SUMMARY

1. An attempt has been made to emphasize and radiographically differentiate the more important pulmonary mycotic infections.

2. Attention has been called to their fundamental similarity.

3. The benign as well as the fatal course pursued by various pulmonary mycotic infections has been discussed.

4. The importance of cooperation between the laboratory, the surgeon, and the roentgenologist in reaching an absolute diagnosis has been mentioned.

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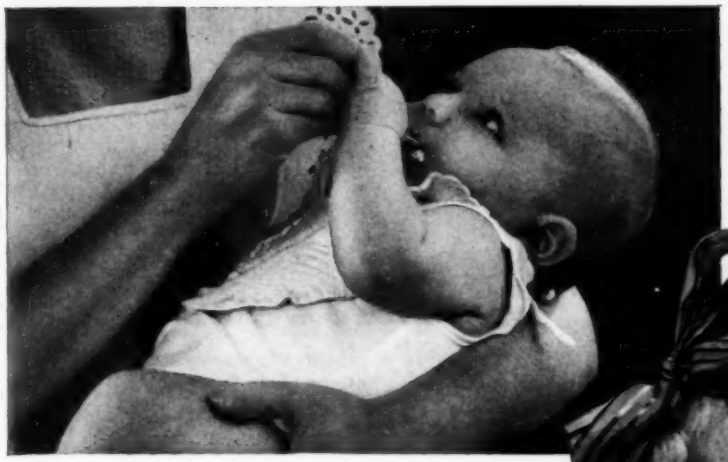

Good Start for a Better Future

GERBER'S 7 STRAINED MEATS

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Each CONVERTIN Tablet is actually two tablets in one:

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Surrounding an enteric-coated core designed to release in the small intestine:

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References: 1. Lee, R. I.: Chicago M. Soc. Bull.: 48:503, 1946. 2. Golob, M.: Am. J. Digest. Dis. 18:308, 1951. 3. McLester, J. S., and Darby, W. J.: Nutrition and Diet in Health and Disease, ed. 6, Philadelphia, W. B. Saunders Company, 1952, pp. 416, 476.

Conventions and Meetings

Announcements

American Osteopathic Association, Fifty-Ninth Annual Convention, Biltmore and Statler Hotels, Los Angeles, July 18-22. Program Chairman, W. Donald Baker, 3450 W. 43rd St., Los Angeles.

American College of Osteopathic Internists, annual meeting, Philadelphia, October 7-10. Secretary, Glennard E. Lahrson, 460 Staten Ave., Oakland 10, Calif.

American College of Osteopathic Obstetricians and Gynecologists, annual meeting, Jacksonville, Fla., February 7-10. Secretary, Harold K. Morgan, 3268 W. 32nd Ave., Denver 11.

American College of Osteopathic Surgeons, annual meeting, Hotel Baker, Dallas, Tex., October 31-November 4. Administrative Secretary, Mrs. O. F. Martin, Box 474, Coral Gables 34, Fla. Program Chairman, J. Donald Sheets, 12561 Third Ave., Highland Park 3, Mich.

American Osteopathic Academy of Orthopedics, annual meeting, Hotel Baker, Dallas, Tex., October 31-November 4. Secretary, J. Paul Leonard, 2673 W. Grand Blvd., Detroit 8. Program Chairman, Walter R. Garard, 809 S. Hobart Blvd., Los Angeles 5.

American Osteopathic College of Anesthesiologists, annual meeting, Hotel Baker, Dallas, Tex., October 31-November 4. Secretary, Crawford M. Esterline, Box 155, Kirksville, Mo. Program Chairman, Armin L. Karbach, 316 N. Center, Arlington, Tex.

American Osteopathic College of Radiology, annual meeting, Hotel Baker, Dallas, Tex., October 31-November 4. Secretary, H. Miles Snyder, Art Centre Hospital, 5435 Woodward Ave., Detroit 2. Program Chairman, John H. Pulker, Flint Osteopathic Hospital, 416 W. Fourth Ave., Flint 4, Mich.

American Osteopathic Hospital Association, annual meeting, Hotel Baker, Dallas, Tex., October 31-November 4. Executive Secretary, Mr. Robert P. Chapman, 1011 Kahl Bldg., 326 Third St., Davenport, Iowa.

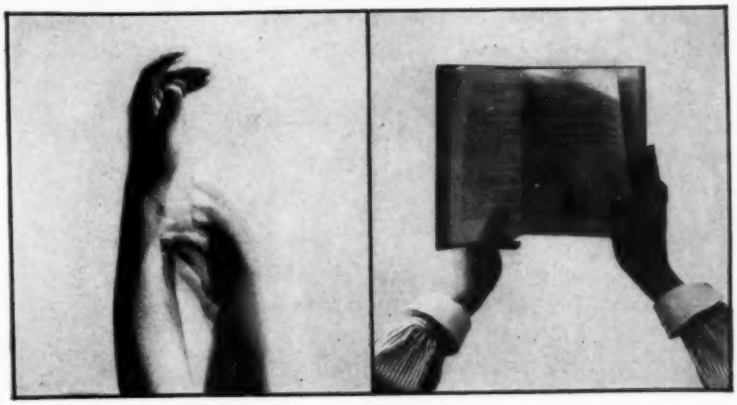
Canada, annual meeting, King Edward Hotel, Toronto, September 16-18. Secretary, Miss Joyce S. Currie, 609 Medical Arts Bldg., Montreal 25. Program Chairman, A. E. Wilkinson, 616 Medical Arts Bldg., Montreal 25.

Central States Society of Proctology, Van Orman Hotel, Fort Wayne, Ind., October 17-19. Secretary, Ralph Deger, 1121 Xenia Ave., Dayton, Ohio.

Colorado: See Rocky Mountain Osteopathic Conference.

Eastern Osteopathic Association, annual meeting, Hotel Statler, New York City, April 2-3. Secretary, Frank B. Tompkins, Baltimore Life Bldg., Baltimore 1. Program Chairman, Chester D. Losee, 212 Prospect St., Westfield, N. J.

Of two patients with poison ivy...



one aggravates the dermatitis venenata by vicious scratching; the result: excoriation and infectious eczematoid dermatitis.

the other is not disturbed by itching. The dermatitis venenata is permitted to clear rapidly and without annoying complications.

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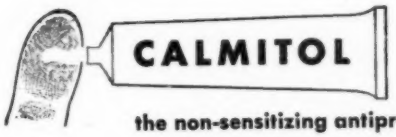

the non-sensitizing antipruritic

1½ oz. tubes and 1 lb. jars

1. Lubowe, I. I.: New York State J. Med. 50:1743, 1950.

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Hawaii, annual meeting, Honolulu, October 25-27. Secretary, Joseph W. Stella, 38 Young Hotel Bldg., Honolulu 9. Program Chairman, C. W. Wyman, 417 National Bldg., Honolulu 13.

Indiana, annual meeting, French Lick Springs Hotel, French Lick Springs, May 14-17. Secretary, Arabelle B. Wolf, 809-13 Odd Fellows Bldg., Indianapolis 4.

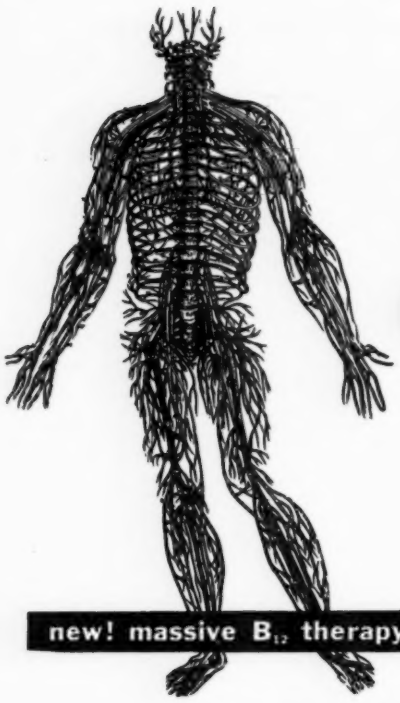
Kentucky, annual meeting, Brown Hotel, Louisville, September 29-30. Secretary, Harold D. Benteen, 2048 Winchester Ave., Ashland. Program Chairman, Martha Garnett, 417 Fincastle Bldg., Louisville 2.

Michigan, annual meeting, Civic Auditorium, Grand Rapids, October 4-6. Secretary, P. Paul Morehouse, 214 S. Superior St., Albion. Program Chairman, B. F. Dickinson, 222 E. Sixth St., Royal Oak.

Missouri, annual meeting, Hotel Colonial and Kentwood Arms Hotel, Springfield, October 13-15. Executive Secretary, Mr. Paul D. Adams, 325 E. McCarty St., Jefferson City. Program Chairman, L. R. Morgan, 3014 S. Main St., Joplin.

New Mexico, annual meeting, Albuquerque, April 28-30. Secretary, Robert E. Smith, 205 N. First St., Lovington.

New York, annual meeting, Hotel Syracuse, Syracuse, October 15-16. Secretary, Robert E. Cole, 417 S. Main St.,



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Geneva. Program Chairman, Allen S. Prescott, 800 Keith Theatre Bldg., Syracuse 2; co-chairman, Francis J. Beall, Jr., 1429 James St., Syracuse 6.

North Carolina, annual meeting, Battery Park Hotel, Asheville, October 21-23. Secretary, S. Dales Foster, 710 Public Service Bldg., Asheville. Program Chairman, Elizabeth E. Smith, 18 Wall St., Asheville.

Oklahoma, annual meeting, Biltmore Hotel, Oklahoma City, November 8-10. Executive Secretary, Mr. Walter L. Gray, 210-212 Braniff Bldg., Oklahoma City. Program Chairman, Fred Green, Alva Osteopathic Hospital, 619 Center St., Alva.

Pennsylvania, annual meeting, Penn Harris Hotel, Harrisburg, September 24-26. Executive Secretary, Mr. George W. Thomas, 510 N. Third St., Harrisburg. Program Chairman, Harry E. Binder, 7247 Charles St., Mayfair, Philadelphia 35.

Rocky Mountain Osteopathic Conference, midyear meeting, Broadmoor Hotel, Colorado Springs, Colo., November 12-14. Secretary, C. Robert Starks, 1459 Ogden St., Denver 18.

Western States Osteopathic Society of Proctology, annual meeting, Shamrock Hotel, Houston, Tex., October 4-6. Secretary, E. F. Walters, 952 E. South Temple St., Salt Lake City 2.

State and National Boards

ALBERTA

Examinations in September at Edmonton. Address Mr. G. B. Taylor, secretary, Medical Board, Office of the Registrar, University of Alberta, Edmonton.

ARIZONA

Those interested in professional examinations should contact Russell Peterson, D.O., secretary, Board of Registration and Examination in Medicine and Surgery, 2747 East McDowell Road, Phoenix.

Basic science examinations September 21 at the University of Arizona, Tucson. Address Herbert D. Rhodes, Ph.D., secretary, Basic Science Board, University of Arizona, Tucson.

ARKANSAS

G. B. Bean, Litttle Rock, has been appointed to the Board of Osteopathic Examiners for a term expiring in May, 1958. L. J. Bell, Helena, and E. M. Sparling, Hot Springs National Park, have been reappointed for terms expiring at the same time.

Officers of the Board are: President, L. J. Bell, Helena; vice-president, P. W. Lecky, El Dorado; secretary-treasurer, E. M. Sparling, Hot Springs National Park.

COLORADO

Basic science examinations in September. Address Esther B. Starks, D.O., secretary, Basic Science Board, 1459 Ogden St., Denver 18.

CONNECTICUT

Basic science examinations October 9 at New Haven. Applications must be filed 2 weeks prior to examination. Address Mr. Herbert S. Harned, secretary, Board of Healing Arts, 52 Whitney Ave., New Haven 10.

DISTRICT OF COLUMBIA

Professional examinations November 8-9. Applications must be filed not later than October 1. Address Mr. Paul Foley, Deputy Director, Department of Occupations and Professions, 1740 Massachusetts Ave., N.W., Washington 6.

Basic science examinations October 18-19. Address Mr. Foley.

FLORIDA

Professional examinations in November. Address R. Philip Coker, D.O., secretary, Board of Osteopathic Medical Examiners, 602 E. 4th St., Panama City.

Basic science examinations in November. Address M. W. Emmel, D.V.M., secretary, Board of Examiners in the Basic Sciences, P.O. Box 340, Gainesville.

IDAHO

Examinations November 9 at Boise. Address D. W. Hughes, D.O., secretary, Board of Osteopathic Examiners, 203 Sun Bldg., Boise.

ILLINOIS

Examinations October 5-7, 160 N. La Salle St., Chicago. Address Mr. Frederic B. Selcke, Superintendent of Regis-

tration, Department of Registration and Education, State House, Springfield.

IOWA

Basic science examinations October 12 at Des Moines. Address Ben H. Peterson, Ph.D., secretary, Board of Basic Science Examiners, Coe College, Cedar Rapids.

MAINE

Examinations November 9-10 at Augusta. Address George F. Noel, D.O., secretary, Board of Osteopathic Examination and Registration, Monument Sq., Dover-Foxcroft.

MARYLAND

Examinations October 16-17 at Baltimore. Applications must be filed 10 days prior to examination. Address Christopher L. Ginn, D.O., secretary, Board of Osteopathic Examiners, 419 N. Charles St., Baltimore 1.

MICHIGAN

Basic science examinations October 8-9 at Detroit and Ann Arbor. Applications must be filed prior to October 1. Address Mrs. Anne Baker, secretary, Board of Examiners in the Basic Sciences, 116 Mason T. Bldg., Lansing.

MINNESOTA

Basic science examinations October 5-6, 101 Wesbrook Hall, University Campus, Minneapolis. Applications must be filed by September 10. Address Raymond N. Bieter, M.D., secretary, Board of Examiners in the Basic Sciences, 105 Millard Hall, University of Minnesota, Minneapolis 14.

MONTANA

Examinations in September. Address Asa Willard, D.O., secretary, Board of Osteopathic Examiners, Wilma Bldg., Missoula.

NEBRASKA

Basic science examinations October 5-6. Address Mr. Husted K. Watson, Director, Bureau of Examining Boards, Department of Health, State Capitol Bldg., Lincoln 9.

NEVADA

Basic science examinations October 5. Address Donald G. Cooney, Ph.D., secretary, Board of Examiners in the Basic Sciences, Box 9005, University Station, Reno.

NEW HAMPSHIRE

Examinations September 8-9 at Concord. Address John S. Wheeler, M.D., secretary, Board of Registration in Medicine, State House, Concord.

NEW JERSEY

Examinations October 19-22 at Trenton. Applications must be filed 20 days prior to examination. Address E. S. Hallinger, M.D., secretary, Board of Medical Examiners, 28 W. State St., Trenton.

NEW MEXICO

Basic science examinations October 17. Address Mrs. Marguerite Cantrell, secretary, Board of Examiners in the Basic Sciences, P.O. Box 1522, Santa Fe.

NEW YORK

Examinations September 21-24 at New York City, Albany, Syracuse, and Buffalo. Applications must be filed not less than 30 days prior to examination. Address Stiles D. Ezell, M.D., secretary, Division of Professional Examinations and Registrations, State Education Dept., 23 S. Pearl St., Albany 7.

NORTH DAKOTA

Leonard W. Mills, Grand Forks, has been appointed to the Board of Osteopathic Examiners for a term expiring in 1956. Gordon L. Hamilton, Minot, was reappointed to the Board for a term expiring in 1957.

OKLAHOMA

Newly elected officers of the Board of Osteopathic Examiners are: President, Ivan E. Penquite, Sapulpa; vice-president, Robert V. Montague, Okmulgee; secretary-treasurer, George R. Thomas, Oklahoma City.

OREGON

Basic science examinations September 11 in Rooms 205 and 206, Portland State Extension Center Bldg., 1620 S. W. Park St., Portland. Applications should be filed not later than August 25. Address Mr. John R. Richards, secretary, Board of Higher Education, Eugene.

RHODE ISLAND

Professional examinations October 7-8. Address Mr. Thomas B. Casey, Administrator of Professional Regulation, 366 State Office Bldg., Providence.

Basic science examinations November 10. Applications must be filed 20 days prior to examination. Address Mr. Casey.

SOUTH CAROLINA

Recently elected officers of the Board of Osteopathic Examiners are: President, Nancy A. Hoselton, Columbia; secretary-treasurer, Ernest A. Johnson, Summerville.

TENNESSEE

Examinations in February and July at Nashville. Applications must be filed 15 days prior to examination. Address M. E. Coy, D.O., secretary, Board of Examination and Registration for Osteopathic Physicians, 1226 Highland, Jackson.

WASHINGTON

H. G. Hofer, Yakima, has been appointed to the Osteopathic Examining Committee.

WEST VIRGINIA

Meeting October 31-November 1 at the Greenbrier Hotel, White Sulphur Springs. Applicants for license by reciprocity will be interviewed. Applications must be filed 30 days before the meeting. Address W. S. Irvin, D.O., secretary, Board of Osteopathy, 202 Broadway, Berkeley Springs.

WISCONSIN

Basic science examinations September 24 at the Assembly Chamber, State Capitol, Madison. Applications must be filed by September 16. Address Mr. William H. Barber, secretary, Board of Examiners in the Basic Sciences, 621 Ransom St., Ripon.

WYOMING

Examinations October 4. Address Franklin D. Yoder, M.D., secretary, Board of Medical Examiners, New State Office Bldg., Cheyenne.

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". . . affords chloral hypnosis without gastric irritation."[1]

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REREGISTRATION OF OSTEOPATHIC LICENSES

Within 60 days after July 1—Indiana, $5.00 for residents, $10.00 for nonresidents. Address C. B. Blakeslee, D.O, secretary, Board of Medical Registration ond Examination, 1000 Kahn Bldg., Indianapolis.

September 1—Ohio, $2.00. Address H. M. Platter, M.D., secretary, State Medical Board, Wyandotte Bldg., Columbus 15.

September—Nebraska, $2.00. Address Mr. Husted K. Watson, director, Bureau of Examining Boards, State Department of Health, Lincoln 9.

October 31—Pennsylvania, Osteopathic Examining Board, $5.00. Address Mrs. Sara H. Longstaff, acting secretary, Board of Osteopathic Examiners, Bureau of Professional Licensing, Harrisburg.

On or before November 1—Missouri, $2.00. Address F. C. Hopkins, D.O., secretary, Board of Osteopathic Registration and Examination, 205 N. Fourth St., Hannibal.

By November 1—Rhode Island, $1.00. Address Mr. Thomas B. Casey, Administrator of Professional Regulation, 366 State Office Bldg., Providence.

EXAMINATION BY NATIONAL BOARD

The National Board of Examiners for Osteopathic Physicians and Surgeons conducts Parts I and II of its examination on the first Thursday and Friday of each May and December at the six approved colleges. Application blanks may be obtained from the secretary or the dean of the college, and the completed application blank, together with a passport photograph and check for the parts to be taken, must be in the secretary's office by the November 1 or April 1 preceding the examination.

Examinations in Part I consist of anatomy, including histology and embryology; physiology; physiological chemistry; general pathology; and bacteriology, including parasitology and immunology.

Part II consists of examinations in surgery, including applied anatomy, surgical pathology, and surgical specialties; obstetrics and gynecology; pediatrics; neurology and psychiatry; public health, including hygiene; medical jurisprudence; osteopathic principles, therapeutics, including pharmacology and materia.

Part III is an oral and practical examination given in Philadelphia, Chicago,

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Beckman[1] remarks, "I think the profession would do well to use this drug more often in insomnia."

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Dosage: SEDATIVE-ANTISPASMODIC, 0.25 Gm. 2 to 4 times daily.

NAUSEA or MOTION SICKNESS: 0.25 Gm., repeated in 30 minutes if necessary. *Hypnosis:* 0.5-1.0 Gm., ½ to 1 hour before retiring. *Contraindicated* only in severe cardiac, hepatic or renal disease.

CLORTRAN is supplied in golden-orange, soft gelatin capsules, 0.25 Gm. (3¾ Gr.) and 0.5 Gm., STABLE CHLOROBUTANOL (7½ Gr.); bottles of 100.

1. Beckman, H.: *Treatment in General Practice* (Saunders) 1948. 2. Rehfuss, M. E., Albrecht, F. K., and Price, A. H.: *Practical Therapeutics* (Williams & Wilkins) 1948. 3. Krantz, J. C., & Carr, C. J.: *The Pharmacologic Principles of Medical Practice* (Williams & Wilkins) 1951.

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Kirksville, and Los Angeles under the supervision of a chief examiner who is a member of the Board and by a panel of associate examiners. Subjects covered in Part III are anatomy; physiology; pathology; osteopathic principles, therapeutics, and pharmacology; surgery; ophthalmology and otorhinolaryngology; obstetrics and gynecology; physical and clinical diagnosis; public health and communicable diseases.

These are oral examinations which the candidate may take after having satisfactorily completed the first 6 months of a 1-year internship in a hospital approved by the American Osteopathic Association for intern training. Part III is given annually at the above-named colleges.

Eligibility requirements are as follows: Part I, satisfactory completion of the first 2 years in an approved school of osteopathy; Part II, satisfactory completion of Part I and of the first two quarters or trimesters of the senior year in an approved osteopathic college; Part III, satisfactory completion of Part II and at least 6 months of a 1-year internship approved by the American Osteopathic Association. The internship requirement does not apply to candidates who took Part I prior to July, 1950.

Application must be filed with the secretary of the Board not less than 30 days prior to the examination dates. Address Paul van B. Allen, D.O., secretary, 1512 N. Delaware Street, Indianapolis 2, Indiana.

THE MAGNITUDE OF THE CANCER PROBLEM*

By Sidney J. Cutler, M.A., and William M. Haenszel, M.A.†

Cancer is the second leading cause of death, ranking next to diseases of the heart. It now accounts for approximately 1 in every 7 deaths in the United States, with an annual toll of approximately 225,000 deaths.

Because of the lack of information from other sources, mortality data, in the past, have been relied on as a measure of the cancer problem. Important as they are, death statistics by themselves cannot define the problem adequately. As therapy becomes increasingly effective, the more curable types of cancer will appear less frequently in mortality records. Furthermore, the medical certification of death cannot be viewed as a complete clinical history. In reporting the sequence of morbid events leading to death, the number of persons dying with cancer is inevitably understated. This can occur because the certifying physician may not know or may neglect to indicate that cancer was present, or because death was obviously due to some cause unconnected with the presence of cancer.

ESTIMATES OF CASES

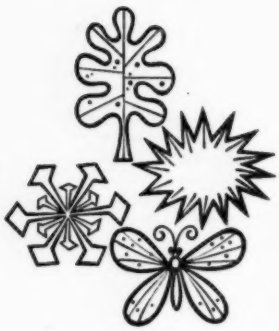
One method of obtaining data on the type and amount of illness in the population is the so-called household survey or family interview. This method is not suitable for cancer studies, and its failings may be attributed to the inability of the respondent to supply the correct information, becauses the physician may not have informed the family of the diagnosis, or because of the respondent's lack of familiarity with technical terms, or his unwillingness to discuss the subject with an interviewer.

The approach adopted by the National Cancer Institute of the Public Health Service has been to canvass all the diagnostic sources within a community—hospitals, clinics, laboratories, practicing physicians—and to obtain names of all persons coming to diagnosis or currently under treatment, so that an unduplicated count of persons ill with the disease might be made. Ten urban areas (Atlanta, Birmingham, Chicago, Dallas, Denver, Detroit, New Orleans, Philadelphia, Pittsburgh, and San Francisco) were surveyed by the Institute in 1937-39 and resurveyed in 1947 and 1948. To insure as complete coverage as possible, data on cancer deaths were obtained at the same time from city and State offices of vital statistics. The mortality records

*Reprinted from *Public Health Reports*, April, 1954.

†Mr. Cutler is an analytical statistician with the Biometry Section in the National Cancer Institute, National Institutes of Health, Public Health Service. Before joining the Public Health Service in November 1948, he was an analytical statistician with the Air Force School of Aviation Medicine (1947-48) and a senior statistician with the New York City Health Department (1946-47).

Mr. Haenszel is head of the Biometry Section of the National Cancer Institute. He was director of the bureau of vital statistics, Connecticut Department of Public Health, for 5 years and before that time a statistician with the New York State Department of Health, in Albany.



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provided information on cancer cases not reported by other sources.

These cancer morbidity surveys have yielded a wealth of information for evaluating the size and nature of the cancer problem. Separate reports have been issued on each of the 10 areas *(1)*, and a summary report is now being prepared. This paper contains a discussion of some of the broad implications of the findings of this series of 10 cancer surveys.

In the 10 cities resurveyed in 1947 and 1948, cancer was diagnosed at an annual rate of 319 new cases per 100,000 population; of every 100,000 persons, 430 were under treatment for the disease sometime during the year. Since the average age of the 10-city population is somewhat younger than for the country as a whole, rates for specific age categories were computed and applied to the age distribution of the United States population in 1950, for the purpose of making national estimates. The incidence rate (new cases per year per 100,000 population) for the United States is estimated to be 328, and the prevalence rate (cases under treatment during the year per 100,000 population) is estimated to be 441. The estimated rates may overstate the true rates for the total United States since current evidence suggests that rates are higher in urban than in rural areas. However, this may compensate for undiagnosed cases for which no allowance has been made.

The age-specific cancer illness rates can also be used for making forecasts. Estimates for the United States of the number of newly diagnosed cancer cases, cases under treatment, and deaths for 1940, 1950, and 1953, and forecasts of these numbers for future years, 1960-75, were made on the assumption that the prevailing age-specific rates will continue. In the 10-year period 1937-47, the age-specific incidence rates increased an average of 14 percent. It is likely that further increases in the rate of cancer incidence will be observed during the next 2 decades, as a result of improved diagnostic and case-finding techniques or increased exposure to carcinogenic hazards. Thus, the projected figures may be considered to be minimal.

Even if the current age-specific incidence, prevalence, and mortality rates were to remain constant, the number of persons affected by cancer will increase by about 50 percent during the next 25 years. This is true for two reasons: The total population of the United States is expected to increase, and the proportion of persons in the older age groups is expected to increase *(2)*. For example, in 1950, 8.2 percent of the population was 65 years of age or older; this proportion is expected to increase to 10.4 percent by 1975. Thus, we see that the expected population trend alone will greatly increase the magnitude of the cancer control problem in the United States, even if the rate of cancer incidence remains constant.

AGE DIFFERENCES IN CANCER INCIDENCE

The incidence of new cases of cancer increases very rapidly with increasing age. For example, the incidence rate is about 40 per 100,000 population at age 25, 475 at age 50, and 1,900 at age 75. There is only one reversal in the increasing rate with increasing age. This occurs at the very young ages. The incidence rate among children under 5 years of age is higher than in children 5 to 14.

We are inclined to emphasize the increase in cancer illness at the older ages, and this is natural, because the rates at the older ages are high. However, when we think in terms of actual numbers of new cases, we must not overlook the very substantial number among children and young adults. For example, roughly 1 of 6 newly diagnosed cases was under 45 years of age. After early childhood, the proportion of new cases in each 5-year age group increases to a peak at 60 to 65 and then decreases. The age at which the greatest number, or proportion, of cases occurs depends on the age distribution of the population from which the cancer cases are drawn. For example, if the population of the United States were older, the peak might occur at ages 65 to 70, whereas if the population were younger, the peak might occur at ages 55 to 60. The cancer incidence rate, however, continues to increase into very old age.

It is important to differentiate between the information provided by a

percentage distribution of newly diagnosed cases and that provided by age-specific incidence rates. The age-specific cancer incidence rates provide a measure of the risk of developing cancer during a specified period of time (1 year, 5 years, and so forth) for persons of a given age or age group. They also serve as an index of the case-finding rate one may expect in different age groups in the population. The age distribution of newly diagnosed cases cannot be used as a measure of risk or rate of case finding, unless we know the age distribution of the population from which these cases came. Approximately 4 percent (3.8 percent) of persons with newly diagnosed cancer were 35-39 years of age. Another 4 percent (4.1 percent) were 80-84 years of age. However, the number of persons in the population who are 35-39 years of age is far greater than the number 80-84 years of age. (In 1950 in the United States, there were 11,246,000 persons 35-39 years of age and 1,059,000 persons 80-84 years of age.) Thus, when the number of diagnosed cases (approximately equal in the 2 age groups) is divided by the number of persons in each age group, the annual cancer incidence rate becomes 148 per 100,000 persons 35-39 years of age compared to a rate of 2,372 per 100,000 persons 80-84 years of age.

Of particular interest is the difference in the lifetime probabilities of developing cancer between male and female white persons. Although the cancer incidence rate for white men is slightly higher than for white women, 31 percent of newborn white male children are expected to develop cancer during their lifetimes compared with 36 percent of newborn white female children. The difference is due primarily to the fact that white women can expect to live an average of 72.4 years, while white men can expect to live only 66.6 years.

In men, the risk of developing cancer of the digestive system is dominant, with a lifetime probability at birth of 10.3 in 100. In women, the risk of developing cancer by age 65 is highest for the genital organs and breast sites. However, the risk of developing digestive cancer after age 65 increases so rapidly that digestive system outranks genital organs and breast as a cancer risk in overall lifetime probability. In both men and women, the probability of developing cancer of the digestive system from age 65 on is very substantial—9.9 per 100 men and 8.4 per 100 women aged 65.

EARLY DIAGNOSIS

Early diagnosis of cancer cases is generally recognized to offer the most hope for successful treatment. This principle of cancer control is supported by data collected through followup of cancer patients. The data are drawn from the cancer register maintained since 1935 by the State of Connecticut *(3)* and from the cancer morbidity surveys conducted by the National Cancer Institute in 10 urban areas, in 1947-48 *(1)*. Each series is based on the experience of more than 40,000 cancer patients.

The chances for survival of cancer cases diagnosed early (while localized at the site of origin) are much better than for cases diagnosed after the tumor has spread to adjacent tissues (regional involvement) or to other organs (remote metastases). For example, of every 100 persons diagnosed as having cancer while localized, 43 lived for at least 5 years after diagnosis, whereas of every 100 persons diagnosed as having cancer after other organs had been invaded only 3 survived 5 years.

In view of the evident benefits of early diagnosis, it is unfortunate that only half of all persons with cancer are being diagnosed while the disease is localized at the site of origin. This record could be improved materially, because half of all cancers develop in organs accessible to direct examination by the physician in his own office. Yet, only 62 percent of cancers developing in accessible sites are discovered in an early stage, and when skin cancers, which generally do not spread to other organs, are excluded, only 50 percent of accessible cancers are diagnosed early. Noteworthy is that in cancer of the breast—a highly accessible site—only 2 of every 5 cases are diagnosed while the cancer is localized. For cancer of the rectum, in both sexes, the proportion is less than half; for cancer of the prostate, one-half; and for cancer of the uterus, the proportion is less than 3 out of 5.

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SUMMARY

The annual number of persons diagnosed with cancer is expected to increase from 530,000 in 1953 to 753,000 in 1975. This estimated increase is based solely on two factors: the forecast increase in the number of persons in the United States, and the forecast increase in the proportion of older people in the population.

Though the figures on the annual number of new cancer cases are impressive, they do not provide an entirely satisfactory measure of the cancer problem. A more satisfactory measure is provided by estimating the probability of developing cancer by a specified age, or during a person's future lifetime. Application of the age-specific cancer incidence rates observed in 10 urban areas in 1947 to the 1950 life table revealed that roughly 50 million people alive in 1950 can expect to develop cancer during their remaining lifetimes, and that one-third of newborn children are expected to develop cancer during their lives. If cancer incidence rates and life expectancy continue to increase, as they have in the past, a substantially larger proportion of the population may be expected to develop cancer. These figures indicate that in terms of its impact on the population of the United States cancer is a major medical problem and promises to be a more serious problem in the future.

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RESEARCH ACTIVITIES IN RHEUMATIC DISEASES*

By Joseph J. Bunim, M.D.†

The rheumatic diseases currently subjected to the most intensive investigation in this country are rheumatic fever, rheumatoid arthritis, gout, and the so-called collagen diseases. The following comments are addressed mainly to the first three of these conditions.

During the past year (July 1952 to July 1953) an estimated total of approximately $1,750,000 was contributed in support of research in the rheumatic diseases in this country. Of this amount, 57 percent was advanced by the Federal Government, mostly by the Public Health Service, and the rest (43 percent) by voluntary agencies, universities, foundations, industrial corporations, and private sources combined.

RHEUMATIC FEVER

The most significant advance made in rheumatic fever as a result of clinical investigation is the demonstration that this disease is preventable. In 1939, it was reported that recurrences of rheumatic fever could be prevented by the continual administration of sulfanilamide to patients who had rheumatic fever. Sulfonamides are effective bacteriostatic agents and suppress the growth of hemolytic streptococci which precede and, it is thought, may incite an attack of rheumatic fever. It was soon discovered, however, that once a streptococcal throat infection developed, administration of sulfonamide was ineffective in preventing the sequel of rheumatic fever. In this regard, penicillin was subsequently found to be decidedly superior to sulfonamides. Although either agent when administered prophylactically to an uninfected person can prevent streptococcal infections and hence recurrences of rheumatic fever, only penicillin can prevent rheumatic fever even when administered after the onset of a hemolytic streptococcal throat infection. This superiority is attributed to bactericidal properties of penicillin in contrast to bacteriostatic action of the sulfonamides. The earlier after onset that penicillin is administered (within 24

*Reprinted from *Public Health Reports*, May, 1954.

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hours) the more effective it will be in preventing rheumatic fever.

It is the practice today, therefore, to keep all rheumatic subjects, especially children and adolescents who are most prone to develop recurrences, on daily sulfonamide or penicillin prophylaxis for 5 years or longer following an attack of rheumatic fever and to administer to all patients who develop a hemolytic streptococcal sore throat large doses of penicillin within the first 24 hours of infection. Under such a regimen only 2 percent of known rheumatic subjects will develop recurrences and 0.2 percent of nonrheumatic subjects will develop rheumatic fever. Without these agents the recurrence rate among rheumatic fever subjects following a hemolytic streptococcal infection has been as high as 50 percent.

During the past 2 years, a new repository type of penicillin (Dibenzyl) has been studied. It shows promise of being a useful compound in simplifying effective prophylaxis of rheumatic fever. It now appears that an intramuscular injection of 1,200,000 units once each month will sustain enough concentration of penicillin in the serum and tissues to prevent hemolytic streptococcal infections and even to eliminate the carrier state.

RHEUMATOID ARTHRITIS

Neither the cause nor the cure of rheumatoid arthritis, the most dreaded of all types of arthritis, is known. Cortisone, hydrocortisone, and corticotropin, when administered to patients who have reversible disease, will very frequently result in dramatic improvement. This may or may not be sustained with prolonged administration. In 80 percent, relapse occurs when the hormone is discontinued. Undesirable effects from this medication are common, and in about 5 to 10 percent they are serious. It is believed that these compounds act by suppressing inflammation. No other adrenal cortical steroid studied thus far has antirheumatic properties. With experience, we have learned to select the proper patients for hormonal therapy more judiciously and to administer these agents more skillfully than in 1949 and 1950.

The National Institute of Arthritis and Metabolic Diseases of the Public Health Service has made available to responsible investigators in this country radioactive C^{14} cortisone acetate and hydrocortisone. Along with other workers the institute is studying the metabolic fate and physiological disposition of these radioactive steroids.

Phenylbutazone is an effective antirheumatic agent, and, by some mechanism of action which is quite different from the adrenal steroids, it too suppresses inflammation. It also has analgesic properties. Phenylbutazone does not alter the course of rheumatoid arthritis, and when it is discontinued the arthritis relapses. Phenylbutazone cannot be

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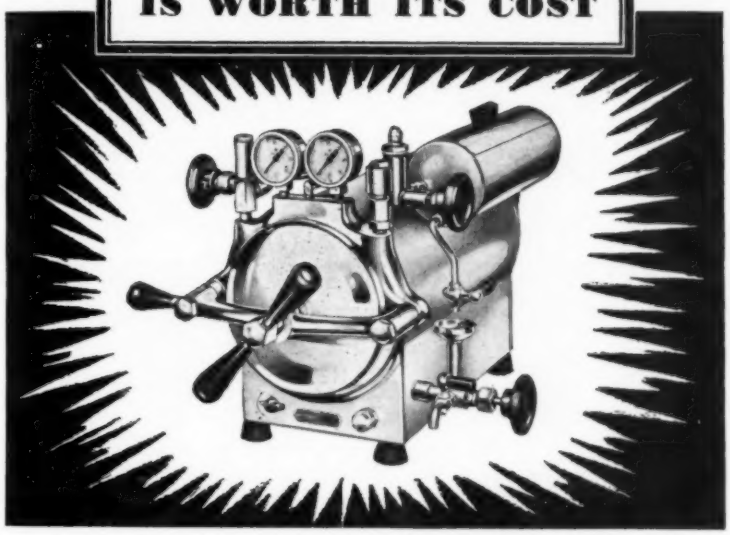

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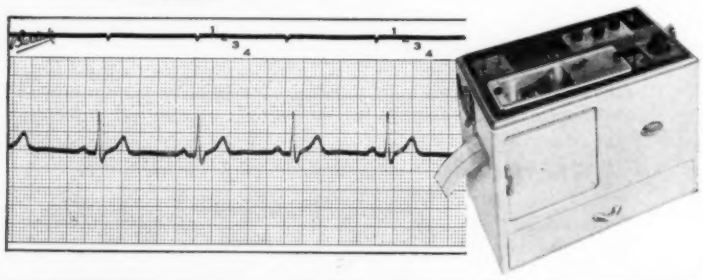

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tolerated by approximately 25 percent of the patients. Some of the toxic effects are serious, and several deaths apparently resulting from its use have been reported in the American medical literature. Although the mortality rate is a small fraction of 1 percent, nevertheless, many physicians have been discouraged from prescribing phenylbutazone because of these fatalities. It is safe to predict therefore that several derivatives chemically related to this compound will be synthesized and tested with the objective of finding a drug which is free from serious toxic effects and yet retains the desirable antirheumatic properties.

Gold compounds, first introduced in the treatment of rheumatoid arthritis 25 years ago, are still commonly used in this country and Europe. The majority but not all of the experienced clinicians in this field believe that there is considerable merit in gold therapy. Gold may help control the inflammatory phase of the disease. It does not repair damaged cartilage or bone, nor does it free an organically ankylosed or fixed joint. Like cortisone, therefore, it is of value only while the arthritis is active. When gold is discontinued relapse occurs in the majority of cases; therefore, many physicians now maintain their patients on gold therapy for years rather than prescribe interrupted courses. The mode of action of gold is unknown.

GOUT

For more than 400 years, colchicine was the only drug known to be of great value in the treatment of gout. In the past 4 years, three important compounds have been accepted as very useful therapeutic agents: corticotropin, phenylbutazone, and probenecid (Benemid).

Although colchicine has the capacity to terminate promptly and dramatically an attack of acute gouty arthritis, it does not increase uric acid excretion nor reduce uric acid level in the blood. In fact, we do not understand how colchicine acts, although it has been used for centuries. Corticotropin can also promptly terminate acute attacks of gouty arthritis. It is superior to colchicine in that the unpleasant gastrointestinal disturbances that appear simultaneously with the therapeutic effect of colchicine do not occur when the corticotropin is used. Although uric acid excretion is increased when this hormone is given, the improvement which follows does not result from this incidental effect.

Phenylbutazone, like corticotropin and colchicine, can terminate an acute attack of gouty arthritis within 24 to 48 hours. It causes a prompt and marked fall in the serum urate level and often but not always a rise in urinary uric acid excretion. Phenylbutazone has certain advantages which makes it more desirable in many cases than either corticotropin or colchicine.

These three drugs are used chiefly to abort acute attacks. For prolonged treatment during the quiescent intervals be-

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tween attacks probenecid, or Benemid, has been found of definite value in gouty patients who have deposits or uric acid tophi in their tissues. Benemid, which is relatively nontoxic, reduces the level of urates in serum and other extracellular fluid from excessive to normal levels by increasing urinary excretion. The daily increase may be as high as 67 percent over premedication level and is accomplished by specifically blocking reabsorption of urates at the renal tubular epithelium. In some cases, it has been demonstrated that Benemid mobilized long-established tophaceous deposits. Some observers have claimed that prolonged Benemid therapy reduces the frequency of acute attacks of gouty arthritis.

Although these new agents make the outlook for the gouty patient more hopeful, they have not given us an insight into the nature of the intrinsic error in purine metabolism which occurs in gout. At the National Institute of Arthritis and Metabolic Diseases and in a few other centers in this country, studies are in progress on intermediary purine metabolism in gouty and nongouty patients using isotopically labelel glycine and uric acid.

AREAS FOR RESEARCH

Important problems that will require thoughtful consideration, careful planning, and sustained scientific pursuit in the immediate future are:

1. To obtain a better understanding of the biochemistry, biophysics, and function of connective tissue which apparently is the primary site of reaction in most of the rheumatic diseases.

2. To elucidate the mechanism of inflammation in these diseases and to develop laboratory methods for satisfactorily screening and evaluating effective anti-inflammatory agents.

3. To determine the role of hypersensitivity or antigen-antibody union in the pathogenesis of certain diseases like rheumatoid arthritis, rheumatic fever, polyarteritis nodosa, and lupus erythematosus.

4. To find a more satisfactory method than is at present available for measuring objectively clinical improvement in arthritis that may result from the administration of a given compound.

5. To collect accurate information on the prevalence and incidence of the various types of arthritis and nonarticular rheumatic disorders in this country.

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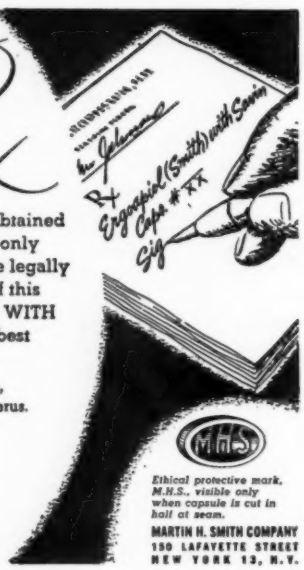

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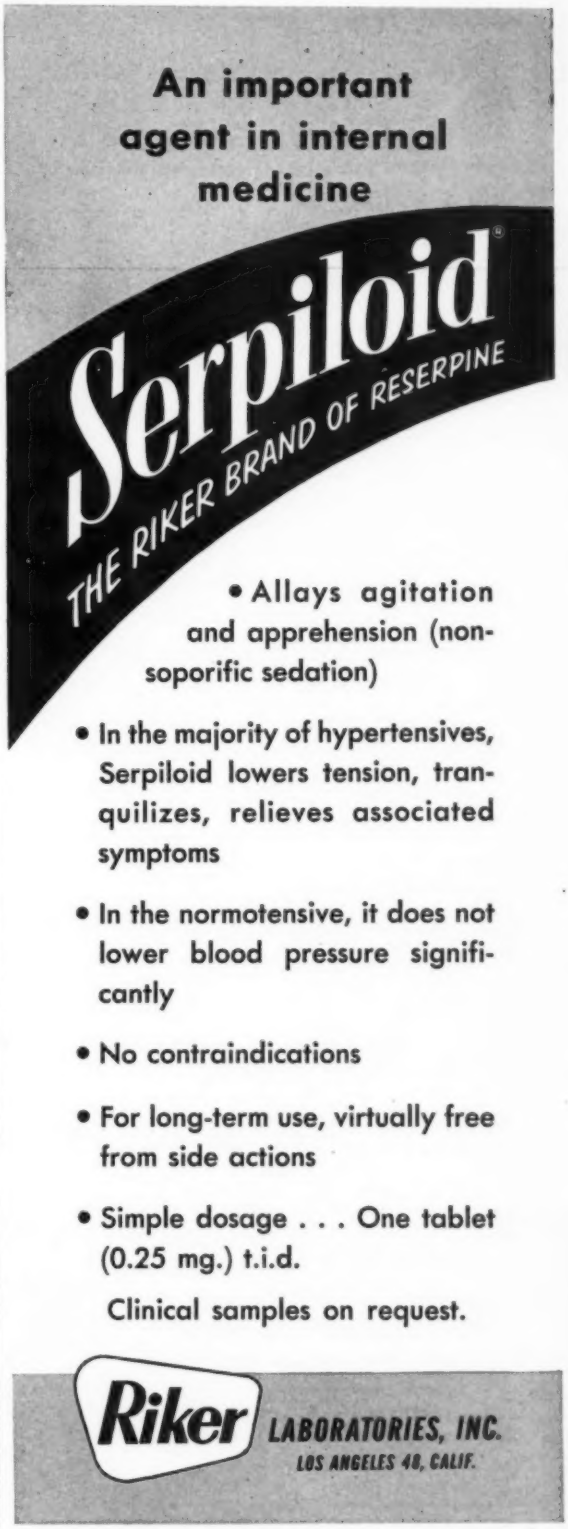
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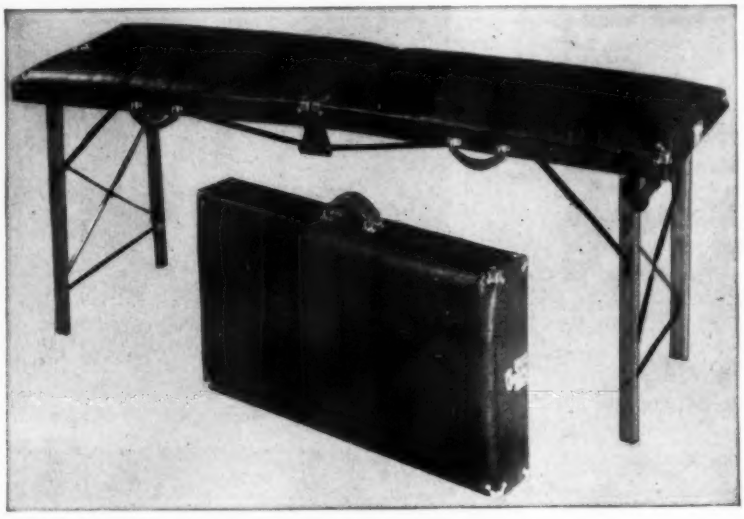

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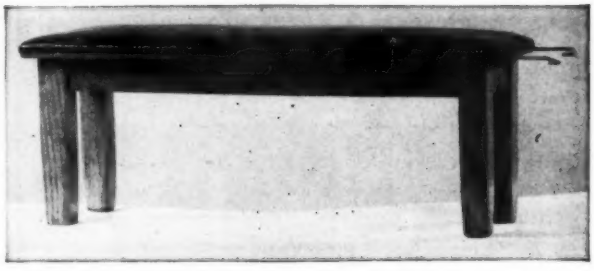

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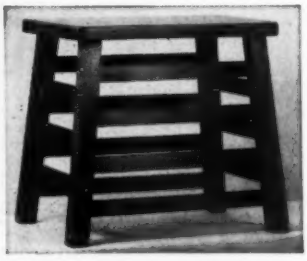

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THE OSTEOPATHIC FOUNDATION

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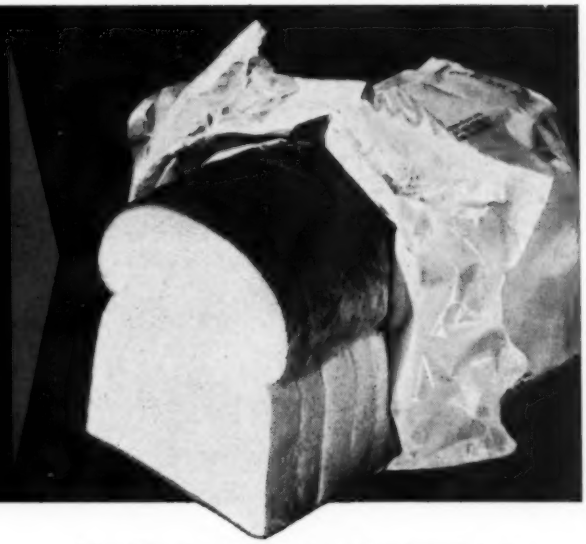
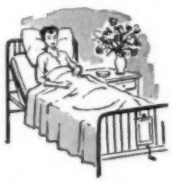
Enriched Bread in *Dietary Planning*



BECAUSE of its nutritional, dietetic, and physiologic values, enriched bread simplifies in many ways the organization of dietaries suited to the special requirements of patients.



FOR THE SURGICAL PATIENT...

The first solid food after surgery is toasted enriched bread, slightly buttered. This practice has become a tradition—almost a ritual—because of the very nature of toast. It is bland, easily digested, and yields little inert residue. Its golden, warm appearance is pleasing to the eye; its mild taste appeals to the palate. Its nutrient energy plays a role in the physiologic and psychologic re-awakening of metabolic processes depressed under the "nothing by mouth" conditions immediately following surgery. With increasing tolerance for food it becomes an important component of the soft diet and later of the therapeutic diet.[1] Its valuable protein, B vitamins, iron, calcium and calories help the patient to regain nutritional efficiency.

FOR THE CONVALESCENT . . .

Enriched bread figures prominently in the dietary regimen in convalescence after acute infections, other serious illness, or trauma.

Supplying 13 grams of high grade protein per 5½ ounces (estimated average daily consumption), enriched bread makes an important contribution to the daily protein need. Its protein, comprising flour, milk, and yeast proteins, functions in the healing of wounds and in the rebuilding of wasted tissues.[2] In addition, 5½ ounces of enriched bread supplies on the average 0.37 mg. of thiamine, 0.23 mg. of riboflavin, 0.34 mg. of niacin, 4.1 mg. of iron, 137 mg. of calcium, and 418 calories.

FOR THE CHRONICALLY ILL...

In the formulation of palatable and nutritious menus for the debilitated, chronically ill, the advantages of enriched bread serve well.

In anorexia, enriched bread or toast stimulates the appetite. It is easily masticated and readily digested, features particularly important for elderly patients. Its favorable textural influence within the alimentary tract[3] promotes good utilization of ingested foods.

---

1. The Committee on Dietetics of the Mayo Clinic: Mayo Clinic Diet Manual, ed. 2, Philadelphia, W. B. Saunders Company, 1954.

2. Sherman, H.C.: Chemistry of Food and Nutrition, ed. 8, New York, The Macmillan Co., 1952, pp. 212, 599.

3. Sherman, H.C.: The Nutritional Improvement of Life, New York, Columbia University Press, 1950, p. 133.

The Seal of Acceptance denotes that the nutritional statements made in this advertisement are acceptable to the Council on Foods and Nutrition of the American Medical Association.

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